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THE
LARYNGOSCOPE.

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RHINOLOGY IN CHILDREN.

Resume of and Comments on the Literature for 1956.*

D. E. S. WISHART, M.D.,
J. B. WHALEY, M.D.,
and
W. B. WALLACE, M.D.,
Toronto, Canada.

This resume of the literature pertaining to rhinology in children once again makes its appearance in the usual form. The journals in the English language, covering the field in otolaryngology, general medicine and pediatrics, have been reviewed. A few headings have been used to classify the articles.

GENERAL ARTICLES ON RHINOLOGY.

The registrar[1] of the Hospital for Sick Children,, Great Ormond Street, London, reported on the effect of prophylactic sulphadimidine in children, subject to recurrent infections of the upper respiratory tracts as follows:

Forty-eight children awaiting tonsillectomy were observed for eight months between August, 1954, and May, 1955. Each child received either prophylactic sulphadimidine or calcium tablets. Half began with 0.5 g. of sulphadimidine daily, while the other half received a calcium lactate tablet

*From the Department of Otolaryngology, Hospital for Sick Children, Toronto, and the Department of Otolaryngology, Faculty of Medicine, University of Toronto.

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identical in taste and appearance. The groups were changed after four months. The trial was conducted on the double blind principle.

Twenty-eight children fared no better on sulphadimidine, three were worse, and in 17 there was no appreciable difference. During the period on sulphadimidine, 25 acute infections were recorded, absence from school totalled 30 weeks, and the family doctor was called to treat 25 illnesses. The corresponding figures for the period on calcium tablets were 60 acute infections, 80 weeks lost from school, and 41 illnesses requiring treatment by the doctor.

Tonsillar hypertrophy and cervical adenitis were favorably influenced by chemoprophylaxis.

Chemoprophylaxis did not seem to affect nasal obstruction or reduce the incidence of the common cold.

Reasons for using chemoprophylaxis in preference to penicillin prophylaxis are discussed.

It is considered that chemoprophylaxis is worth a trial as an alternative to tonsillectomy in children subject to recurrent infections of the upper respiratory tract and, if instituted promptly, would result in a reduction of the number requiring this operation.

Wickes,[2] while admitting the conclusions of Burke, maintains that before accepting these as a permanent gain, it is necessary to look at the long term aspects. He asks, "Why do some children develop so many upper respiratory infections in the first year or two at school?" and answers, "presumably because they do not develop an immunity as rapidly and as effectively, as their fellow pupils. They must succumb to more infections before they become protected."

If this is true, then chemoprophylaxis is probably only postponing infections rather than inducing them.

If we wish to use modern antibacterial agents for the long-term benefit of the patient, we shall have to obtain more information about the after-behavior of these children. It might (or might not) be found that in subsequent winters,

a treated group developed the infections which chemoprophylaxsis had previously spared them, whereas, an untreated group had by then grown out of this phase; in other words, that treatment had reduced the intensity but increased the duration of this tiresome period—the age group when so many children are referred, in desperation, for tonsillectomy, when all that is needed in most cases is patience.

Craddock[3] considers that Burke's paper gives scientific backing to a method of reducing the number of children requiring tonsillectomy. This number can be still further reduced if the natural history of the acquisition of resistance to upper respiratory infections is taken into account.

A child who has only one or two attacks of tonsillitis in one winter, following several the previous winter, should be allowed a further 12 months of observation before tonsillectomy is performed, unless there have been complications, such as severe or recurrent otitis media. It this practice were generally followed the large waiting lists for T's and A's would be pruned still further.

Goodman,[4] commenting on Burke's paper, finds fault with the figures given in one of her tables, because he believes that sulphadimidine in the dosage given could hardly be called effective in reducing the incidence of upper respiratory infection in children to a satisfactory level.

Burke[5] agreed with certain commentators on her paper that most children outgrow their suspectibility to infections of the respiratory tract and this, in her opinion, is the main justification for giving prophylactic chemotherapy during the winter months for one or two years, to those subject to frequent infections. She carefully replies to Goodman's criticisms.

To the question: "What measures are worth taking in the home to protect young children against frequent colds?" the *British Medical Journal*[6] made reply as follows:

"Any immunity conferred by infection with the common cold virus seems to last for only two or three weeks, and there is no known way of increasing this by vaccine or other ther-

apy. There is no good evidence that susceptibility to the virus is increased by lack of vitamins or other therapy. There is no known evidence, in spite of current advertisements, that extra vitamins, vaccines, drugs, or intraviolet light therapy help in the slightest to protect against colds. A good mixed diet with an adequate vitamin content is, of course, necessary for the maintenance of optimum health.

The only effective way of preventing colds is to avoid them by keeping children away from contact with others. This is clearly impracticable, but it is reasonable to keep a child who is prone to severe colds, away from parties and crowded shops and vehicles, at least, for one or two winter seasons. Plenty of fresh air and good ventilation are always indicated for children.

Parrott[7] reviewed certain recent advances in our knowledge of respiratory diseases of non-bacterial origin. These have been a perennial problem to those dealing with illnesses of children, military recruits and industrial workers.

The adenoidal-pharyngeal-conjunctival viruses have been abbreviated into the name APC viruses, and there are at least ten immunologically distinct types.

Serological studies show that 50 per cent of infants in the 6 to 11-month age group shows evidence of having been infected with at least one type of APC virus. There is a sharp rise in evidence of infection from ages 1 to 2, and a less prominent rise from ages 3 to 5. Gradually thereafter there is increasing evidence of infection with at least one of the virus strains.

Clinically, not enough data is available concerning most of the APC virus types, but the four major clinical features are fever, conjunctivitis, pharyngitis and posterior cervical lymphadenopathy. Pharyngo-conjunctival fever occurs epidemically and sporadically. It affects all ages although children are most susceptible. Antibiotics and chemotherapy have not been effective treatment.

There are many questions still unanswered. The most provocative question is: What are the possibilities of immun-

izing human beings against these very prevalent viruses? From information now accumulating, it is not too unlikely that a vaccine might be developed which would eliminate a significant amount of respiratory disease.

An epidemic of pharyngo-conjunctival fever occurring in a summer camp for boys is reported by Sobel and her coworkers.[8] The outbreak was due to the APC Type III virus and happened during the summer of 1955. The clinical picture of pharyngo-conjunctival fever, or APC fever, is characterized by conjunctivitis, pharyngitis, cervical adenitis and spiking fever of four or five days' duration. The disease primarily affects children and is usually epidemic in nature.

The camp under discussion had 250 boys in attendance, and they were divided into a junior and senior camp. There were two outbreaks; one in July, and one in August, the latter being the main one, as it involved 62 per cent of the campers. A very careful and extensive study was made of APC fever in this localized epidemic, and it is well reported in the article. The characteristic findings of spiking fever for a few days, unilateral or bilateral follicular conjunctivitis, follicular hypertrophy of the anterior pillars of the tonsils, enlargement of the nasopharyngeal lymphoid tissue, and cervical adenitis were all recorded. Less common findings were generalized glandular enlargement, hepatomegaly, splenomegaly and meningismus.

A heavy growth of APC III virus was isolated from the conjunctivae, throat and stools of eight out of ten patients studied. There was a significant rise of complement fixing antibodies in the convalescent sera. The disease appeared to be spread by droplet infection, and chemotherapy was ineffective.

Torticollis resulting from upper respiratory infections, particularly sinusitis, is discussed by Brown[9] from cases he has seen over the past 25 years. Acute wry neck is not an uncommon symptom in older children. The sternocleidomastoid muscle on one side is in painful spasm and the patient holds the neck rigid in the position of least pain. The head may be inclined to the affected side, or more often to the

opposite side. On the affected side, the deep cervical nodes are almost invariably enlarged and tender. Sinusitis is the usual cause of this acquired torticollis. In almost every case of torticollis, tenderness of the sinuses and the cervical nodes is greater on the side of the involved sternocleidomastoid muscle. Following treatment of the sinusitis, all three inflamed areas decrease in tenderness. In some cases, tonsillitis or pharyngitis may be present as well.

The treatment of acquired torticollis is directed at the cause of the infectious process present. Penicillin is given intramuscularly in single doses of 300,000 units daily for about three days. Isotonic astringent nose drops are instilled three times daily, or an inhaler may be used. This is followed by the application of hot wet towels to the face for five to ten minutes. The head should be elevated at night and during the day, the child is placed in a sitting posture or reclining slightly. At night, the sternocleidomastoid muscle may be relaxing by inclining the head to the affected side, supported by an extra small pillow under the neck and face. Under this regime, relief is usually obtained in a day or two.

Boyes, Jones and Miller[10] report seven cases of primary tuberculous infections in the mouth. This type of infection is uncommon, yet tuberculous submandibular adenitis is not uncommon. These seven cases all presented the combination of a painless tuberculous ulcer in the mouth and a regional tuberculous lymphadenitis. A history of trauma was present in five of the group, and in four of these it was dental extraction. The primary lesions, following dental extraction, were at the site of the tooth socket or in the sulcus between the jaw and the cheek. The lesions varied in size from a small circular ulcer to one over an inch long. Granulation tissue also was found in some of the dental sockets, and these each had alveolar abscesses. Two children without a history of injury had a large painless ulcer on the gum above normal intact teeth. In each case the gland swelling brought the patient to the family doctor.

It seems that any chronic lesion in the mouth, any area of dental trauma which fails to heal completely, or any area of

granulation tissue is under suspicion, and should be examined histologically. If there is also a swelling of the glands, the possibility of primary tuberculosis must always be considered.

ACUTE SORE THROAT.

Chapple et al.[11] made a clinical and bacteriological survey of cases of acute sore throat in general practice, and described the results of a strictly controlled trial of oral penicillin, sulphadimidine, and a placebo in its treatment.

Whereas 61 per cent of patients receiving the placebo were still ill on the third day of treatment, the corresponding rates for those on sulphadimidine and penicillin, were 38 per cent and 31 per cent respectively. The difference between 61 per cent and the other two rates, was statistically significant, but that between 38 per cent and 31 per cent was not; however, the difference between penicillin and sulphadimidine in those ten years of age and over, was greater than in those under ten years. Fewer failures of treatment occurred in those who received penicillin.

The unexpected observation was made that the results of treatment were practically the same in streptococcal and non-streptococcal sore throats. Analysis of the presenting symptoms and signs in patients with and without hemolytic streptococci in pre-treatment throat swabs showed only minor differences between the two groups. No clue to the etiology of the non-streptococcal illnesses was found, apart from their apparent response to both sulphadimidine and penicillin.

The British Medical Journal[12] states that five years ago it published the results of two studies of the efficacy of sulphonamides in the treatment of acute sore throat: one concluded that these drugs had no effect; the other could identify no effect by observations on the duration of individual signs and symptoms, but gained an impression from the patient's appearance that the tratment was of some benefit.

It draws attention to the trial, embracing 308 patients, reported by Chapple et al., and especially to the fact that only 45 per cent of all these patients were found to have hemolytic streptococci in their throats, the cause of the infection in the

remainder being unknown. In commenting on the similar findings of the two earlier studies referred to, it had written: "Acute sore throat is one of the commonest ailments, and if half of the patients suffering from it are being attacked by a micro-organism which has hitherto escaped identification, this is probably the widest field in the whole domain of microbic infection which remains to be successfully explored." If this was true five years ago, it evidently is true even today. The APC viruses may reasonably be suspected of causing infections in young children which are unresponsive to chemotherapy, but if the beneficial effects of sulphonamides and penicillin are accepted as proved, these viruses cannot be responsible in older patients, in view of the fact that they have been isolated in tissue culture from adenoids in a medium containing 100 units of penicillin per ml. Responsiveness to both forms of chemotherapy employed in the trial by Chapple et al. argues a bacterial cause, and it should be worth while again to study the ordinary flora of the throats of similar patients with a more open mind. It is not long since that bacterium coli was regarded as no more than saprophytic in the intestine, but several types of this species are now recognized as pathogenic. It could be, for instance, that among the highly heterogeneous alphahemolytic and non-hemolytic streptococci which are now casually dismissed as commensals, there are in fact varieties which can cause an acute catarrh.

Bailey[13] for some years, has found a sulphathiazole lozenge the most constantly effective agent in most sore throats. He uses a 0.325 g. pleasantly flavored tablet, produced by Eli Lilly, and for adults, direct that one should be dissolved slowly in the mouth every half hour for six hours, and every hour thereafter.

Adams[14] commented as follows: "The problem of the unknown infective agent in this condition, which remains open in the St. Paul's Cray survey, (Journal, March 31, p. 705) and is commented on in your annotation (Journal, April 7, p. 793) is also a problem in acute tonsillitis, where there are similar difficulties in identifying an organism which can be regarded as pathogenic in about the same proportion of cases.

"In the search for the hitherto undiscovered invasive element in the acute sore throat, it may be of some significance that the infection in acute tonsillitis is bismuth sensitive, as most patients recover in 24 hours after receiving a small dose of bismuth by intramuscular injection, or more conveniently in the hospital by rectal suppository. The sensitivity to bismuth in acute tonsillitis suggests that this may also occur in the acute sore throat, and it would seem an experiment well worth undertaking to ascertain its effect in a clinical controlled trial in this condition; and it also suggests laboratory studies to carry out further investigations of the antibiotic spectrum of bismuth against bacterial and virus cultures."

Florman and Bergher[15] bring to the attention of those concerned with the newborn infant, a benign condition of the pharynx which may mimic serious bacterial or viral infections. This benign pharyngeal erythema and follicle formation in the newborn was first noticed in the nursery during a routine examination of an infant on the third day of life. It was described as "four or five whitish-yellow papulovesicles on the tonsillar pillars and soft palate, typical of the herpangina seen on older children." The characteristic lesion was seen on one or both of the anterior pillars. One to three slightly elevated, discrete white to yellowish circular areas, about 1 mm. in diameter, were seen on an erythematous base. These might have increased by size and number by the following day. In one or two days the elevated areas had disappeared and only a general erythema remained.

Sometimes the elevated areas were replaced by shallow ulcers. Approximately three days after the lesions were first observed, the pharynx appeared to be normal. The temperature, feeding habits, weight curve and stool pattern all were normal, as were leucocyte counts. About 20 per cent of the babies in the nursery were found to have these pharyngeal lesions. Bacterial cultures from the throats, produced the normal flora found in the neonatal period. Viral studies also were negative. Pharyngeal smears were made for cytological examination, and the cells suggested the possibility that the pharynx of the newborn might be a "target area"

for estrogen withdrawal effect. None of these infants appeared to be ill.

The treatment of children with acute streptococcal pharyngitis with a single penicillin dose, is reported by Hartenstein and Feldman.[16] This report is concerned with the results obtained in the treatment of 12 patients. Their ages ranged from 3 to 11 years, and they presented themselvs for diagnosis and therapy at various periods after the onset of their illnesses. No history or evidence was present of previous rheumatic fever, rheumatic heart disease, or acute glomerulonephritis. Throat and nose cultures were taken, and serological identification of the streptococci was carried out. In 10 of the 12 patients, the pharyngitis was found to be due to Group A streptococci.

Treatment consisted of a single dose of repository penicillin product (Panbiotic), which contained 600,000 units of benzathine penicillin G, 300,000 units of procaine penicillin G, and 300,000 units of buffered potassium penicillin G. The 1,200,000 units of this penicillin mixture was in an aqueous suspension of 2 ml. volumes, and administered as an intramuscular injection.

All patients with symptoms felt better within one to two days, and the throats usually appeared normal within three days. No allergic reactions to penicillin were observed during this study. All patients developed a slight induration and tenderness at the site of the injection, but this disappeared completely in less than a week. Neither acute rheumatic fever nor acute glamerulonephritis was noted in any patient during the period of observation.

A new syndrome in an unusual case of retropharyngeal abscess is presented by Townsend.[17] The retropharyngeal space is subject to bacterial invasion from the nasopharynx, and from the cervical vertebrae, but since the advent of chemotherapy abscesses of this space have become rare. As the pyogenic organisms acquire resistance to therapeutic agents, the severe complications of nasopharyngitis, once controlled, are recurring, and often present new and unusual signs and symptoms.

The case reported was that of a five-year-old girl, with a chief complaint of a stiff neck of one month's duration. Originally she was seen because of a sore throat, "stiff neck" and cervical adenitis. She received 300,000 units of procaine penicillin G, on each of two successive days with a concurrent subsidence of temperature and pharyngeal symptoms, but the "stiff neck" remained. Two weeks later, she complained of inability to swallow solid food. About two weeks later again, she was referred to hospital, and X-rays were taken of the cervical region. Examination of the patient showed a chronically ill child in no acute distress, who was unable to swallow solid food. Her neck was held semi-flexed. Flexion of the neck was painless, but extension even to the central position, was not possible. Pain also limited lateral movement of the head to 15 degrees in either direction. A tender gibbus posteriorly at the level of C-2 was present. Complete examination of the oronasopharynx was not possible, but with this limitation, the remainder of the physical examination was normal. The white blood count was 35,000 with 80 per cent polymorphonuclear leucocytes. The X-ray film showed the presence of a large retro-pharyngeal mass, obliterating the normal cervical lordosis, and subluxation of C-2 on C-3.

Laryngoscopy without anesthesia, revealed a yellow apex to the mass directly opposite the glottis. This was incised and approximately 30 cc. of thick, yellow pus, under tension, was evacuated. On the second postoperative day she was able to move her neck with minimal evidence of the previous limitation, and an X-ray showed a marked reduction in the size of the retro-pharyngeal space. A later X-ray showed complete restoration of the normal anatomic relationship of the cervical elements, and no evidence of scar or abnormality of the cervical vertebrae.

TONSILLECTOMY.

To the questions, "what conditions in children make tonsillectomy an urgent operation, and what conditions justify the inclusion of a child on a long waiting list for tonsillectomy," *The British Medical Journal* made the following reply[18]:

"Removal of the tonsils and adenoids may be considered urgent if there is persistent gross obstruction of the upper respiratory passages, or if there is recurrent otitis media or otorrhea associated with infection of the tonsils and adenoids. Tuberculous cervical adenitis, particularly if there is marked enlargement of the glands as a result of associated pyogenic infection, is another indication for early operation.

"There is still controversy about the need to remove tonsils and adenoids for less urgent reasons, some holding that eventual spontaneous recovery is likely to occur without operation. On the other hand, a period of a few years of persistent ill-health and of recurrent attacks of acute tonsillitis may be avoided by operation, and obviously in that case the earlier this can be performed the better, but it is still worth while even if the waiting-list is long. Indications are lesser degrees of persistent upper respiratory infection and obstruction and frequent recurrences of acute tonsillitis. A less obvious indication is the combination of general ill-health with poor appetite, failure to gain weight, restlessness at night, together with evidence of upper respiratory infection, such as postnasal catarrh, an irritating cough, and enlargement of the tonsillar glands.

"The prolonged delay which so often occurs before operation is very unsatisfactory, but even if this amounts to a year, it is found that need for operation remains unchanged in the great majority of cases. Under ideal circumstances this might no longer be true, but usually operation forms an effective short cut to health in those cases in which the indications have been properly assessed."

Fry[19] writes that for the past five years he has kept records of the incidence of the various infections of the upper respiratory tract in children, and has found that, whatever the true cause of them, they tend to run a characteristic natural course. He finds that if we wait long enough, most children will lose their coughs and colds spontaneously and naturally, without interference of the ear, nose and throat surgeon. His own tonsillectomy records have been only 5 per cent over the past ten years, and in spite of this his child patients seem to grow into healthy adolescents.

Librach[20] states that the crux of the tonsillectomy problem, is that hypertrophy of the tonsils is a *normal* physiological process occurring in children between five and ten years old. There are then, only two logical indications for operation: *1.* chronic tonsillar infection, and *2.* obstruction of nasal breathing.

Burkinshaw[21] replies to a critic by quoting from M.R.C. Special Report No. 227 a summing up of the whole matter, which he considers excellent and fair: "Few today would encourage the retention of diseased or obstructive tonsils, or would cast doubt on the real value of the operation in properly selected cases as demonstrated above; but it is a little difficult to believe that among the mass of tonsillectomies performed today (1938) all subjects for operation are selected with true discrimination, and one cannot avoid the conclusion that there is a tendency for the operation to be performed as a routine prophylactic ritual for no particular reason, and with no particular result."

He adds the observation that the unnecessary tonsillectomy still seems to be unnecessary, and that necessary tonsillectomy still seems to be necessary.

Librach[22] cannot let the contention in M.R.C. Report No. 227 go unanswered: "This implies that tonsillectomy may render the patient at least as resistant as non-tonsillectomized persons, and that the case against operation is statistically unanswerable. Those of us working in infectious diseases hospitals have ample proof (if this be needed) that tonsillectomy *per se*, does not influence resistance to infections to any considerable degree. I can provide many instances, including my own personal experience, when the patient has contracted nearly every common infection since operation."

He believes that unnecessary tonsillectomy removes a valuable link in the immune chain. The operation is likely to influence subsequent events beneficially only when the tonsils themselves are diseased.

Collins and Granatelli[23] reported observations on anesthesia for 50 children undergoing tonsillectomy. All were

induced in vinyl ether-diethyl ether sequence by the open drop method. Thereafter anesthesia was maintained with an ether oxygen mixture. In 25 patients the anesthesia was maintained by ether hook insufflation, and in the other 25 an endotracheal tube was used.

In this study a second anesthetist was present to make observations every two or three minutes, and record cardiovascular respiratory and oxygen saturation changes. The presence or absence of blood post-operatively in the trachea was determined.

They recommend the endotracheal method as causing less asphixial phenomena, allowing better control of respirations, better working conditions for the surgeon, and minimizing the trouble caused by bleeding and secretions. The authors stress the importance of proper psychological preparation of each child, and also favor the use of preoperative medication with rectal Seconal and intramuscular scopolamine.

Comment:

Park,[37] reporting from the Hospital for Sick Children in Toronto, recently presented a paper on 25,000 tonsillectomy anesthesias without complications. His recommendations were very simple: *1.* Proper psychological preparation; *2.* The majority of his cases received no preoperative sedation. This allows for rapid return of vigorous cough and gag reflexes; *3.* Anesthesia was by ethyl chloride induction and ether oxygen insufflation. A sand bag under the shoulders, with head low, will prevent the aspiration of blood and secretions; *4.* No child was allowed to leave the operating table until vigorous cough reflex had retuned. This was a remarkable series, because many of the operations were performed by otolaryngological housemen working with anesthetists, who were in training as well.

Engelking[24] reviewed the literature on the problem of post-tonsillectomy poliomyelitis and made an analysis of 888 cases of poliomyelitis and 2,813 tonsillectomies from a district in the Southwest United States from 1949 to 1953. A survey of the literature, he found, permits one to believe as he

chooses; that removal of tonsils increases the susceptibility to polio for years, if not for life; or conversely, that the loss of tonsils no more influences the chance of developing poliomyelitis than does the loss of the appendix.

The literature is very confused, but it seems safe to say that those who believe in a causal relationship have based their opinions on experimental evidence, isolated cases, small series of cases, or series studied without due regard to statistical methods. On the other hand, those who deny the relationship have investigated larger series, and have used sound statistical methods. They have also gone into a side of the picture previously ignored, namely, the incidence of poliomyelitis following large series of tonsillectomies.

The investigation of 888 cases of poliomyelitis and of 2,813 adenotonsillectomies performed over the same five-year period, confirmed the opinion that there is no causal relationship between tonsillectomy and post-tonsillectomy poliomyelitis. It also showed clearly the fallacies and pitfalls of basing conclusions upon single years and small series of cases from small institutions.

The solution to the problem is the application of common sense to the individual case. When poliomyelitis is prevalent, no elective surgery should be performed. When it is not prevalent, tonsillectomy may be safely performed in the cases in which it is indicated.

The author feels that the best way to settle the problem in the light of present knowledge would be for some national body to undertake a controlled study to determine what happens after tonsillectomy, as well as how many tonsillectomized patients develop poliomyelitis.

ADENOIDS.

Guggenheim,[25] in two papers, reviewed the historical background of direct adenoidectomy. Among others, he mentioned the contributions of Yankauer, Lothrop, Love and L. K. Guggenheim. These techniques have evolved as the result

of long continued efforts to solve one of the most difficult and generally neglected problems in our specialty.

In the second paper, he pointed out that while even a poorly done adenoidectomy will usually relieve postnasal obstruction, such incomplete surgery will frequently fail to prevent recurrent otitis media, or alleviate conduction hearing loss due to obstructed Eustachian tubal orifices.

The importance of removing lateral lymphoid curtains was stressed. Strong direct illumination gives the best results, and ideally one should have general anesthesia deep enough to abolish pharyngeal reflexes, and relax the soft palate.

He favors the Love palate retractor, and prefers sharp dissection for removal of remaining lymphoid tissue after preliminary removal of the main adenoid mass with the adenotome. Attention to obtaining strict hemostasis between the various steps is important, so that proper visualization of the nasopharynx is possible. For hemostasis he uses gauze sponges, sparingly moistened with 1:1000 adrenalin, and held with gentle pressure each time for a period of three to five minutes.

The worthwhileness of the method can be interpreted only in terms of results, and he promises a later paper which will consider the problem in these terms.

Johnson[26] described a method to control adenoid hemorrhage, using a Foley catheter. The catheter, a 30 cc. capacity size, is inserted through one side of the nose, and is then partially inflated and is drawn into the adenoid area to control the bleeding. The advantages are mainly its simplicity and lack of discomfort to the patient, especially at the time of removal.

Adams[27] reported some studies in Eustachian deafness, and its response to nasopharyngeal irradiation. He found tubal dysfunction to be a common condition in childhood, and the commonest cause of social and educational deafness with a small, but appreciable risk of long term deafness.

The negative middle-ear pressures were shown always to

recover spontaneously in time, but the hearing losses in some cases are the result of secondary changes interfering with the round and oval windows in some cases; in others, due to inhibition of cochlear function. There was no indication that the deafness was other than acquired, and recovery occurred in such a large proportion that it was evident that in most cases the pathology is reversible.

Ninety per cent were successfully treated by careful removal of tonsils and adenoids. In this series the remaining 83 cases were treated by irradiation with recovery in 63 of the number. Throughout the survey the criterion for the presence of tubo-tympanic dysfunction was impaired mobility of the intact M. T. by Seigle. In earlier years this was a visual estimate, but since 1952 the Tomanometer was used and the middle-ear negative pressures recorded in mm. of mercury.

A persistently raised negative middle-ear pressure, even without material deafness, should be regarded as a potential auditory risk which requires active treatment. The first measure is a meticulous tonsil and adenoid operation. If this fails, irradiation has proved effective in 75 per cent of the cases.

DRUGS.

A new nasal decongestant, Tyzine (tetrahydrozoline hydrochloride) was introduced into therapy late in 1954, after extensive animal experimental studies and clinical trials. Soon after its introduction, a depressant effect upon the central nervous system was noted, particularly in children of less than one year of age. Chasid, Lehr and Levy[28] report an alarming reaction in an infant, following the nasal instillation of tetrahydrozoline hydrochloride. This 10-week-old infant was admitted to hospital, because of a severe central nervous system depression, manifested by stupor and shallow breathing of 14 hours' duration, culminating in apnea and cyanosis. This episode occurred after the mother had administered tetrahydrozoline hydrochloride nasal solution. The baby had a "cold" and nasal discharge, and was not doing

well under usual treatment. The physician, therefore, prescribed tetrahydrozoline hydrochloride, 0.1 per cent in a plastic squeeze bottle. The mother instilled one drop into each nostril from the inverted bottle, and two hours later, the child was noted to be unusually lethargic and to have stertorous respirations. Two hours before admission to hospital, the physician found the baby flaccid, apneic and severely cyanotic. As an emergency measure, he administered oxygen under positive pressure, adrenalin and Cortisone. Soon afterwards the baby was hospitalized, showing very little improvement. The child was kept in an oxygen tent for 24 hours, and his respirations gradually improved, and he became more alert. Additional therapy included penicillin, streptomycin and vitamin supplementation. On the fourth hospital day, the baby was discharged, recovered.

In discussing this case, the authors point out that parents should be made aware of the dangers of unsupervised medication, especially in infants, and should be warned to discontinue the drug at the first sign of depressant reaction; also, the use of spray bottles for medication in infants should be discouraged.

Brainerd and Olmsted[20] report three cases showing extreme drowsiness as a toxic effect of Tyzine hydrochloride, and the experimental results of nasal instillation of this decongestant in 19 other infants. The three cases admitted to hospital all showed lethargy and shallow respirations. Each had had a "cold" previously, and Tyzine nose drops had been prescribed. The ages were two months, ten weeks and six weeks. They all recovered readily in a few hours by the administration of oxygen and "sleeping off" the drug.

In the experimental group, the procedure was to instill Tyzine 0.1 per cent nasal solution in doses of one to four drops every four hours, for a period of one to four days. The infants were observed carefully at the time of administration of the drops, for signs of drowsiness, tremulousness, change in color or respiration, feeding behavior, vomiting and signs of nasal irritation. One group of six infants was given one drop in each nostril every four hours. A second group of six

infants was given a course of two drops in each nostril every four hours, and a third group was given three drops every four hours. Five infants were tested with the intranasal administration of four drops every four hours. Some infants were given more than one test period, making a toal of 25 experiments. From these tests marked drowsiness was noted in four cases. In six instances moderate drowsiness was present and 15 infants exhibited no significant drowsiness.

In addition to the infants studied, five rabbits were given Tyzine 0.1 per cent intranasally, in doses of one to five drops in each nostril three times daily for a period of five days. None of the rabbits exhibited any significant drowsiness or other changes suggestive of toxicity during the test period.

CONGENITAL ABNORMALITIES.

Poch-Vinals and Calvo[30] reported a case of unilateral choanal atresia in a 12-year-old girl, which they treated successfully by a transpalatine surgical approach. They have described in detail, the embryology of the area involved, but the exact origin of these atresias is still somewhat unclear. In the main three surgical approaches have been tried. Transnasal, transinus (through the antrum) and transpalatine approach is used.

The transpalatine approach has further advantages: *1.* The operation is done under direct vision; *2.* permanency of the opening is made possible by removal of the posterior border of the septum and the base of the atresia (the adjacent hard palate); *3.* a flap of membrane is preserved to cover the raw surface.

Cracovaner and Goodman[31] presented a case of congenital bilateral choanal atresia treated by the transpalatine approach, and discussed the embryology anatomy and diagnosis of this condition. Most authors feel that these atresias are the result of failure of the bucconasal membrane to rupture; some, however, believe that it may be caused by an osseous projection from the horizontal portion of the palatine bone.

The definitive choana is bounded medially by the posterior border of the vomer and the nasal crest of the palatine bone.

Above are the ala of the vomer and the sphenoid bone. Laterally the medial plate of the pterygoid process of the sphenoid plus the perpendicular plate of the palatine bone. Below is the horizontal plate of the palatine bone.

Bilateral atresia in the newborn, is a medical emergency. The salient features are recurrent cyanosis, inability to nurse, and excessive nasal discharge.

The attendant must constantly try to keep the infant's mouth open, because he cannot learn to do this without aid. This is due to the fact that most infants' instinct is to breathe through the nose. Occasionally a tracheotomy is needed. Feeding has to be done by gavage. Diagnosis is confirmed by catheters or probes, and by X-rays using radiopaque media. The authors treated their case by the transpalatine approach with good result still being maintained after two years.

Congenital aglossia is reported upon by Fulford, Ardean and Kemp.[32] They reviewed several cases from the literature and gave a case report of their own. The embryological formation of the tongue is a complex process, and the cases studied were all interrupted at an early stage. The anterior part of the tongue was absent, and the posterior part was represented by a rudiment. The case reported was a boy aged five weeks, who was admitted to the hospital because he was not taking his food well, and was losing weight. Examination showed a fairly well nourished baby with a receding lower jaw. The mucosa of the floor of the mouth was filiform, and appeared to consist of normal tongue mucosa, but there was no tongue. The mucosa was raised laterally into two ridges which ran alongside the medial borders of the mandible, and these folds probably contained muscle, as they moved when the baby cried. In the midline of the posterior part of the floor of the mouth, was a mobile pyramidal projection about one-half cm. in height. The hard and soft palates, uvula, tonsils and larynx were normal. The only other abnormality was syndactily of the left hand.

On feeding from a bottle he swallowed slowly, but without choking or nasal regurgitation. He breathed and cried normally. An excellent cineradiographic investigation is in-

cluded by the authors, and this showed the way in which the baby feeds and swallows. At three months, the baby was feeding normally, and had no trouble afterwards up to ten months, when he was last examined. Apparently the disability caused by congenital aglossia is not very great after the first three months.

TUMORS.

MacNeil[33] reports a case of extranasal and intranasal tumor in a newborn child. A tumor about the size of a cherry was present over the bridge of the nose in the midline. It was firm and did not fluctuate, and was only slightly moveable. The nasal root was not widened, nor did the baby have any rhinorrhea. Roentgen studies neither confirmed nor ruled out the existence of any communication from the cranial cavity to the tumor. Diagnostic puncture of the tumor was negative.

The tumor was removed next day under local anesthesia, through a midline vertical incision. No difficulty was encountered in shelling the tumor out; there was no sign of a pedicle attaching it to any location. It was firm in consistency, and had a smooth greyish-red surface covered with a thin fibrous capsule. Pathological diagnosis—ganglioglioma.

Three weeks after the baby's discharge from the hospital, the mother reported that the swelling had recurred, and that the baby appeared to have difficulty with its breathing. Examination showed that recurrence was taking place, and in addition a greyish red tumor was seen in the left nasal cavity, and appeared to be covered by septal mucous membrane. It was obviously either an extension from the external tumor, or another tumor originating from some other location. Roentgen studies showed that the soft tissue mass adjacent to the left side of the nose, did not communicate with the interior of the cranium.

When the baby was six months old, a second operation was performed. An inverted U-incision was made over the top of the nasal bridge to include the external tumor, which was then dissected out and found to be similar to and about the

same size as the original. The incision was extended and the cavity of the nose entered by reflecting the nasal bone with the soft tissues attached, bringing the intranasal tumor into good view: The latter was dissected out and removed. No deformity or opening into the cranial cavity was found. There is no doubt that a defect was present at birth, but it must be assumed that it subsequently closed. Pathological diagnosis: Recurrent nasal glioma. The condition has remained cured.

Ramon Del Villar[34] described three cases of extranasal tumors of neural tissue. These tumors are so rare that no definite theory as to their origin has been established.

The cases described appeared to be benign congenital tumors of firm consistency, which were located at one side of the root of the nose. There was little tendency toward growth and no tendency to metastasis or recurrence. They are rather hard, of reddish or reddish-purple coloring and were easily displaceable from the deeper layers. They could readily be confused with dermoid cysts or hemangiomas. The diagnosis is not likely to be made except after surgical intervention and histological examination.

The occurrence of brain tissue outside the cranial cavity is extremely rare. When it is found in the nose and throat it is a congenital anomaly, which probably represents completely or partially severed remnants of the anterior cerebral vesicle. Three cases are described by Low, Scheinberg and Anderson,[35] which were found to have brain tissue in the nose and throat. The first case was a seven-week-old infant with a growth on the bridge of the nose which had been present since birth. It had enlarged about three times since birth to become equal in size with the nose, but did not impair nasal breathing. The tumor was removed via an elliptical incision across the nose. A portion of the nasal bone and triangular cartilage, to which the growth was attached, was also removed.

The second case was a seven-year-old Puerto Rican girl with a growth on the nose. She had been born with a mass on the bridge of the nose, and this had been removed at the

age of four days. The tumor recurred about three years previously, and had been growing gradually since. The patient had had seizures manifested by clonic movements, since the age of four years. The tumor was removed surgically, and a portion of it was found to have eroded through the upper medial part of the maxillary bone at its junction with the frontal and nasal bones, and seemed to extend up under the cribriform plate. The seizures continued, and further investigation including pneumo-encephalography, showed that the tumor had an anatomical connection with the brain, and that other cerebral anomalies coexisted.

The third patient was a three-year-old boy who was admitted for tonsillectomy. He had a cleft in the posterior part of the soft palate, 1.5 cm. to the left of the midline. The tonsils were enlarged, and the posterior tonsillar pillar on the left was absent. The tonsils and adenoids were removed, and at operation a submucosal, firm, fibrotic mass was found presenting in the left nasopharynx. This mass was removed also, and the child made an uneventful recovery.

Pathological examination showed all the three tumors to be made up of glia and a network of connective tissue. The authors point out that when a tumor is encountered near the nose or in the pharynx, this anomaly of brain tissue should be kept in mind, because of the occasional connection with the brain, or meninges, and the consequent risk of meningitis.

Acuna[36] wrote about nasopharyngeal fibroma, and observed that it is a disease that attacks young persons and is also known as a juvenile fibroma, and basal fibroma or fibroangioma of the nasopharynx. These cases are frequent in Mexico. The General Hospital in Mexico City has seen 70 cases in the last six years. Spontaneous regression has been observed only once in 50 years, so that it is unwise to delay treatment in hope of such regression. The General Hospital in Mexico City has achieved a record of 100 per cent success during the past five years, most patients having been treated surgically.

REFERENCES.

1. Burke, J. B.: Prophylactic Sulphadimidine in Children Subject to Recurrent Infections of the Upper Respiratory Tract. *Brit. Med. Jour.*, 538, March, 1956.

2. Correspondence: The Catarrhal Child. *Brit. Med. Jour.*, 746, March, 1956.

3. Correspondence: The Catarrhal Child. *Brit. Med. Jour.*, 746, March, 1956.

4. Correspondence: The Catarrhal Child. *Brit. Med. Jour.*, 861, April, 1956.

5. Correspondence: The Catarrhal Child. *Brit. Med. Jour.*, 1046, May, 1956.

6. Questions and Comments: Prevention of Colds in Young Children. *Brit. Med. Jour.*, 184, January, 1956.

7. Parrott, Robert H.: APC Viruses in Respiratory Diseases; Clinical Aspects. The Laryngoscope, p. 628, June, 1956.

8. Sobel, G.; Aronson, B.; Aronson, S., and Walker, D.: Pharyngo-Conjunctival Fever, Report of an Epidemic Outbreak. *Jour. Dis. Child.*, 92:596, Dec., 1956.

9. Brown, E. E.: Torticollis Resulting from Upper Respiratory Infections, Particularly Sinusitis. *Arch. Pediat.*, 73:382, Oct., 1956.

10. Boyes, J.; Jones, J. D. T., and Miller, F. J. W.: The Recognition of Primary Tuberculous Infection of the Mouth. *Arch. Dis. Child.*, 31:81, April, 1956.

11. Chapple, P. A. L.; Franklin, L. M.; Paulette, J. D.; Tuckman, E.: Woodall, J. T.; Tomlinson, A. J. H., and McDonald, J. C.: Treatment of Acute Sore Throat in General Practice. *Brit. Med. Jour.*, 705, March, 1956.

12. Editorial: Treatment of Acute Sore Throat. *Brit. Med. Jour.*, 793, April, 1956.

13. Correspondence: Acute Sore Throat. *Brit. Med. Jour.*, 921, April, 1956.

14. Correspondence: Acute Sore Throat. *Brit. Med. Jour.*, 981, April, 1956.

15. Florman, A. L., and Bergher, M.: Benign Pharyngeal Erythema and Follicle Formation in the Newborn. *Amer. Jour. Dis. Child.*, 91:549, 1956.

16. Hartenstein, H., and Feldman, H.: Treatment of Children With Acute Streptococcal Pharyngitis With a Single Penicillin Dose. *Jour. Pediat.*, 48:318, March, 1956.

17. Townsend, E. H., Jr.: Retropharyngeal Abscess. Presentation of a New Syndrome in an Unusual Case. *Jour. Dis. Child.*, 92:308, Sept., 1956.

18. Any Questions: Indications for Tonsillectomy. *Brit. Med. Jour.*, 936, April, 1956.

19. Correspondence: Is Tonsillectomy Necessary? *Brit. Med. Jour.*, 302, Aug., 1956.

20. Correspondence: Is Tonsillectomy Necessary? *Brit. Med. Jour.*, 549, Sept., 1956.

21. Correspondence: Is Tonsillectomy Necessary? *Brit. Med. Jour.*, 713, Sept., 1956.

22. Correspondence: Is Tonsillectomy Necessary? *Brit. Med. Jour.*, 827, Oct., 1956.

23. Collins, V. J., and Granatelli, A.: Anesthesia for Tonsillectomy in Children. *J.A.M.A.*, p. 5, May 5, 1956.

24. Engelking, Charles F.: The Problem of Post-Tonsillectomy Poliomyelitis. The Laryngoscope, p. 1541, Dec., 1956.

25. Guggenheim, Paul: Direct Adenoidectomy. *I. A. Historicoliterary Review.* II Technical Aspects. *A.M.A. Arch. Otolaryngol.*, p. 178, Sept., 1956, and p. 361, Nov., 1956.

26. Johnson, F.: The Control of Adenoid Hemorrhage with a Foley Catheter (Balloon Type). *A.M.A. Arch. Otolaryngol.*, p. 295, Mar., 1956.

27. Adams, W. Stirk: Studies in Eustachian Deafness and Its Response to Nasopharyngeal Irradiation. *Jour. Laryngol. and Otol.*, p. 512, Sept., 1956.

28. Chasid, E.; Lehr, D., and Levy, W.: Severe Respiratory Depression Following Nasal Instillation of Tetrahydrozoline Hydrochloride. *Jour. Pediat.*, 48:66, Jan., 1956.

29. Brainerd, W. K., and Olmsted, R. W.: Toxicity Due to the Use of Tyzine Hydrochloride. *Jour. Pediat.*, 48:157, Feb., 1956.

30. Poch-Vinals, R., and Calvo, R. Ramos.: Choanal Atresia. *A.M.A.* Arch. Otolaryngol., p. 559, June, 1956.

31. Cracovaner, Arthur J., and Goodman, Merrill: Congenital Bilateral Choanal Atresia. *A.M.A. Arch. Otolaryngol.*, p. 267, Oct., 1956.

32. Fulford, G. E., Ardean, G. M., and Kemp, F. H.: Aglossia Congenita. *Arch. Dis. in Child.*, 31:400, Oct., 1956.

33. MacNeil, F. A.: Intranasal Meningocele. *Canad. Med. Assn. Jour.*, Jan., 1956.

34. Del Villar, Ramon: Astrocytomas of the Nose. *A.M.A. Arch.* Otolaryngol., p. 466, May, 1956.

35. Low, N. L.; Scheinberg, L., and Andersen, D. H.: Brain Tissue in the Nose and Throat. *Jour. Pediat.*, 18:254, Aug., 1956.

36. Acuna, Ricardo T.: The Nasopharyngeal Fibroma and Its Treatment. *A.M.A. Arch. Otolaryngol.*, p. 451, Dec., 1956.

37. Park, N. S.: Anesthesia for Tonsillectomy and Adenoidectomy in Children. Paper presented before Ont. Med. Assn., Toronto, May 30, 1957.

DIAGNOSING AND TREATING CONGENITAL FISTULA OF THE AURICLE.*

Report of Two Cases.

WILLIAM SKOKAN, M.D.,

Fort Worth, Tex.,

When Irish missionaries tried to Christianize the pagan Dutch of Holland, they were strongly resisted by the Freislanders, who would not accept the new faith. The Irish missionaries, marking the pagans to distinguish them from the Christians, punctured their auricles. Some people still believe that families in which the pitted ears are found, are descendants of the pagan Dutch.[53]

The pitted ear—congenital fistula of the auricle—is a comparatively common but an easily overlooked developmental anomaly. It is mentioned infrequently in medical literature, not because it is rare, but because it seldom produces disturbances.[36] The condition is also known as fistula auris congenita, congenital auris fistulae, congenital auricular sinuses and congenital auricular fossa.[49] Since information about this condition is scarce in most textbooks, some physicians may be unaware of its existence. As a result, they err in its diagnosis and subsequently undertake improper treatment.[34]

These pits may be pointed out, occasionally to the patient's surprise. The fistula is sometimes covered with a crust. When the crust is removed, a drop of seropurulent fluid is exposed, although it is so inconspicious that many persons are happily unaware of its presence.[14] The fistulas may be mistaken for sebaceous cysts, abscesses, dermoids, blackheads, fungus and tuberculosis. In one patient to be discussed, the fistula was mistaken for furunculosis of the external auditory canal; it was initially a marginal helicine fistula

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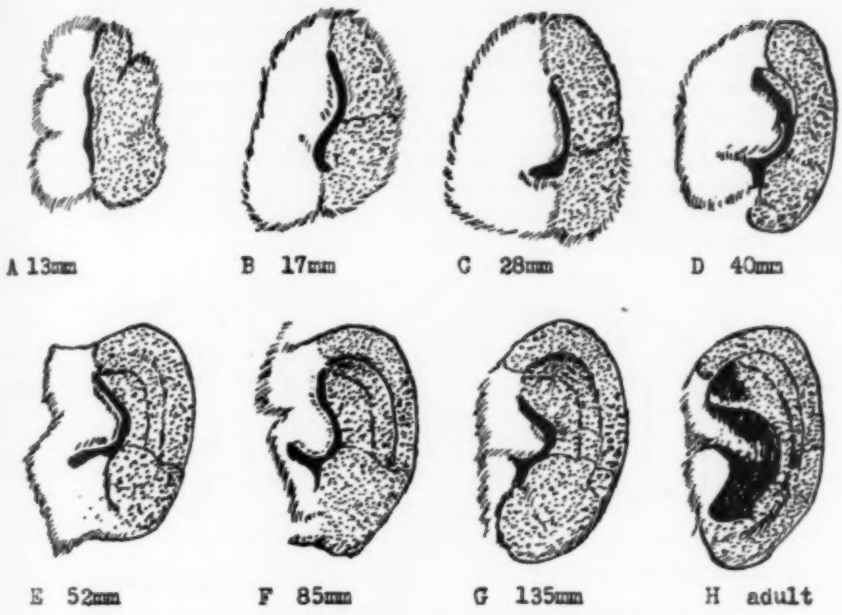

Fig. 1. Stages in development of external ear. (After Streeter, Carnegie Cont. to Emb., Vol. 14, 1922.) The parts derived from the mandibular side of the cleft are unshaded; the parts from the hyoid side are indicated by stippling and by irregularly placed dashes.

which extended into the ear canal as a result of recurrent infection. The second patient had a pit below the left tragus. It was associated with mild hemiatrophy of the left side of the face and a recurrent infection with abscess formation back of the left ear lobe. The pathologic condition back of the ear was found to be a tract paralleling the whole length of the external auditory canal (see Fig. 10). Mastoiditis was at one time considered to be the causative factor.

Present day books say that the external ear is developed from the upper part and the boundaries of the first external pharyngeal (hyomandibular) groove.[6] Six eminences appear on the mandibular and hyoid margins of the first external

groove. The various parts of the auricle are formed from the eminences on the two arches, and from the skin immediately posterior to the eminences in the hyoid arch. The exact part played by the individual eminences is a matter of some doubt.

Failure of the tubercles about the hyomandibular cleft to fuse with each other or failure of some of these tubercles (hillocks) to grow normally may produce a variety of malformations.[40]

Fig. 2. After Streeter. Showing the relative location of the hillocks. Hillock 6' goes into formation of the antitragus.

The tympanic membrane is formed in the bottom of the external auditory meatus in the position of a "separating membrane" between the first pharyngeal pouch and the first external groove.[6]

Some interesting work by embryologists will be reviewed to show the somewhat doubtful origin of the auricle. The origin of ear pits was and still is a matter of considerable debate.

In 1877, Moldenhauer,[35] studying the development of the external ear of the chick embryo, noticed the occurrence of two pairs of hillocks in the first and second branchial arches.

His[21,22,23] described the tubercles, or hillocks, around the first gill cleft in a 12 to 30 mm. human embryo. He numbered them from one to six. Of the two hillocks found on the mandibular arch, the lower one develops into the tragus and the upper becomes the spina helicus of the helix. One hillock at the upper limit of the first gill cleft forms the rest of the

helix. On the hyoid bar His found three hillocks. The upper one becomes the antihelix, the next lower one the antitragus. The lowest one seems to become covered in by the tragus and later appears to form the lobe. So in essence the tragus comes from the first hillock, the helix comes from the union of hillocks 2 and 3, the antihelix comes from the fourth, and the antitragus comes from the fifth and the lobule from the sixth (see Fig. 1). His seemed to think that marginal sinuses

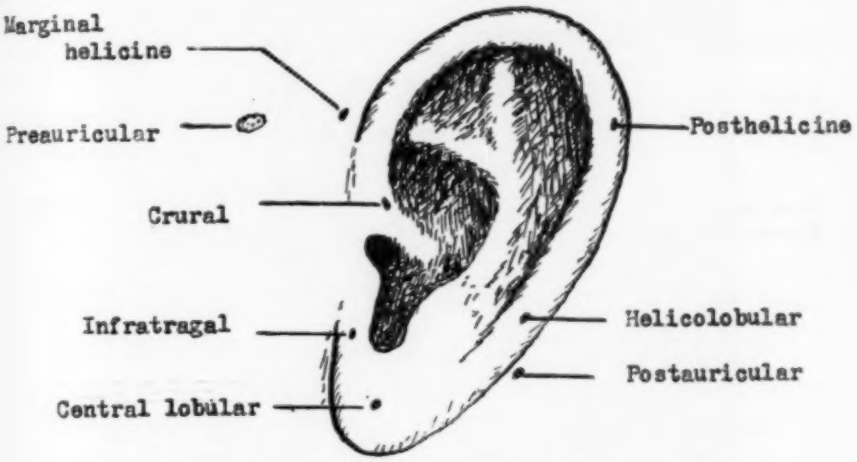

Fig. 3. Diagram showing the various locations of the congential auris fistulae. The term infratragal is used to describe Case 2.

were accentuations of a groove related to the first two tubercles (hillocks).

Streeter[54] discussed a modification in naming six the 6′ hillocks. He suggested that hillock 5 disappears early, that 6′ becomes the antitragus and that hillock 6 is involved in forming the ear lobule (see Fig. 2).

Gradenigo[18] in 1888 in mamalian study, found six hillocks, but contended that they do not form the auricle proper, which instead is formed from elevations of the hyo-mandibular bars. These elevations appear at about the same time as the

hillocks are flattening out. The auricle would appear to be formed from a fold of skin resembling an eyelid, and is situated behind hillocks 4 and 5.

Schwalbe[45,46] in 1891 had described the hillocks as did His, but he brought out the significant fact that reptiles, which lack external ears, have very distinct hillocks in the embryo, very much as do animals that develop auricles. In 1897 he[47,48] again described the hillocks substantially as His did, and he

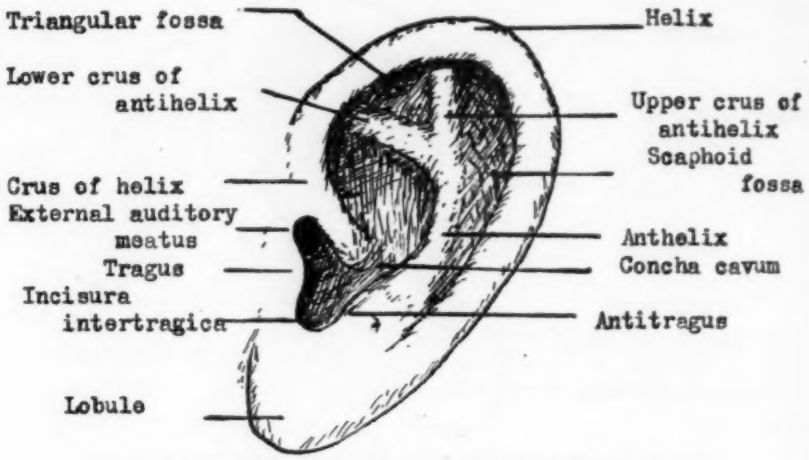

Fig. 4. Diagram showing various parts of the auricle.

agrees with Gradenigo that the fold behind hillocks 4 and 5 seems to be the origin of the auricles.

Streeter[54] concluded that until more concrete evidence is obtained the hillocks must be regarded as transitory and as incidental rather than fundamental to the development of the auricle. The histologic change which inaugurates the formation of the auricle consists of a proliferation and condensation of the mesenchyme. Accompanied by evidence of marked activity of the ectoderm over the whole auricular area, more growth takes place over the whole surface of the hyoid bar than over the posterior half of the mandibular bar (see Fig.

Fig. 5. Showing the sutures in place. Note lack of inflammation.

1). It is interesting that the mandibular derivatives are relatively very large in the earlier stages, as are the derivatives of the lower end of the hyoid bar. The hillocks seem merely to be foci in which mesenchymal proliferation is temporarily most rapid.

The hillock theory of His seems the most uniformly accepted of proposed theories. Jones and Wen,[25] have questioned whether the fistulas are caused by faulty union. In the first place, the distribution by no means appears to follow the lines of union of the hillocks; in the second place, the

hillocks themselves are of such low elevation that it is hardly conceivable that the pit or fistula is formed at their junction site. Jones and Wen did acknowledge that the tragus appears to be the only mandibular derivative of the pinna, and that the line of the first pharyngeal depression appears to coincide with the line of election for the pre-auricular fistula.

Fig. 6. Sutures were removed just prior to photograph.

Stammers[51] agreed that the fistulas were not remnants of the first branchial cleft but result from aberrant coalescence of the six tubercles which His described. He also disregarded the so-called macrostoma theory by stating that in its severest forms the macrostoma never extends as far back as the ear. The pits, therefore, could not be formed because

Fig. 7. The posterior lesion at time of picture was quiescent.

of macrostomal tendency. Keith[26] believed fistulas to be formed by poor fusion of tubercles.

Congdon[8] stated that the crural, marginal helicine and preauricular fistulas are derived from the groove between hillocks 2 and 3. Adult position results from skin migration. The posterior helicine fistula develops between hillocks 4 and 5, the helicolobular fistula between 5 and 6, the central lobular between 6 and 6′. He also asserted that data on normal development of posterior auricular sinuses are lacking and that their origin remains unsolved.

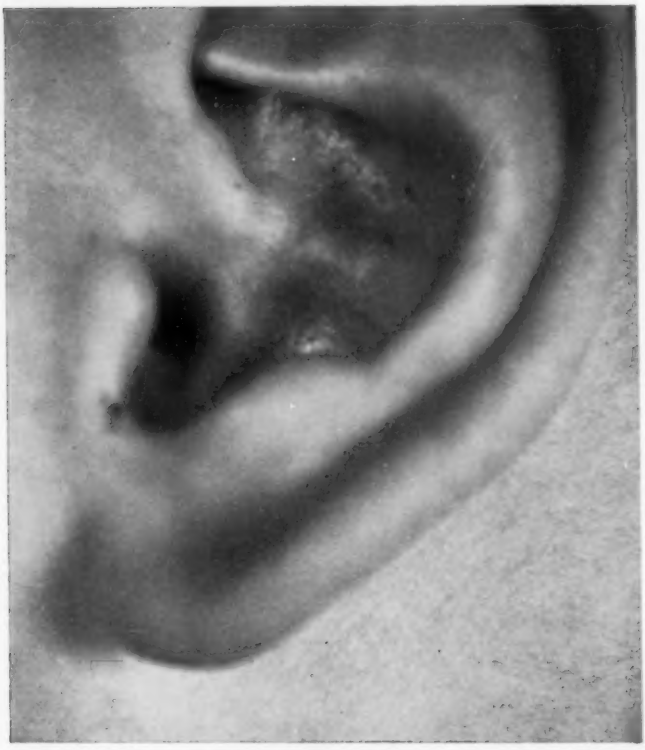

Fig. 8. Infratragal congenital auris fistula. Note fullness in the floor of the external auditory canal.

As has been shown, the fistulas are classified by their location, Fig. 3 is for purpose of orientation. I have designated the site below the tragus as infratragal. The origin of the infratragal fistula might be logically due to the failure of hillocks 1 and 6 to close. Anatomy of external ear is shown in Fig. 4 for purpose of review.

The controversy over the origin of the congenital fistulas of the auricle continues, however, for even as late as in 1930 Fedrici[15] sought to establish the origin of the marginal helicine sinuses from the dorsal end of the first branchial groove.

Fig. 9. Fistula after removal.

Becker and Brunschwig[3] in one case showed columnar epithelium in the blind pouch of the sinus tract. To them, this demonstrated the branchial cleft origin of the lesion.

Onodi[38] in 1918 showed that branchial fistulas have been seen to open into the external auditory canal and never seem to connect with the auris fistulae.

Virchow's[56] patient in 1864 showed a fistulous opening in the external ear canal which communicated with a fistula,

Fig. 10-A. Tract below the external auditory canal, demonstrated by iodized oil.

supposedly an auris fistula; however, now it is thought to have been a vestige of a branchial groove.

Kipp[28] noted that there was no connection between the auris fistulae and the external auditory canal or the tympanic cavity. Among all the hundred of depressions seen, nobody has ever found a branchial cleft derivative connect with a congenital auris fistulae.

Ballantyne[2] in 1904 stated that fistulas apparently never

Fig. 10-B. Tract below the external auditory canal, demonstrated by iodized oil.

open into the auditory canal. Schwabach[44] made the same observation in 1879.

Heusinger[19] reported the first case in 1864; the description fits the marginal helicine type. Both the left and right sides showed small holes ahead of the ascending helix. The same patient also had a little hole in the left side of his neck. Heusinger described a small skin elevation in which each of the ear pits was located.

In contradistinction to Heusinger, Urbantschitsch[55] in 1878 found no elevation of the skin surrounding the ear pit.

Paget[39] first presented cases in England in 1877. His case presentation stated that the pinnae of the ears were peculiar in shape, being somewhat triangular; and that on the upper part of each helix, close to the skin of the temple, was a small depression large enough to admit a No. 4 shot. The patient's grandmother and her eldest daughter had similar ear pits in similar locations. Deafness was not prevalent in the patient. Paget implied that these were traces of branchial fissures of the helix. Doran[11] presented a three-year-old child with a marginal fistula a quarter inch deep.

Kipp[28] presented the first cases in the United States in 1880. He presented six patients, four in males and two in females. The canals ranged from 3 to 10 mm. in depth, and all were the marginal type. No abnormalities of the auricle were noted, hearing was normal, and there was no connection between the fistula and the external auditory canal or of the tympanic cavity. Three patients had cysts along with the fistulas.

Incidence varies in different races. Selkirk[50] showed an incidence of .9 per cent in 3,660 white children and an incidence of 5.2 per cent in 346 negro children. The ratio of males to females is 1 to 4 in both races.

Congdon[8] in his study of Oriental races found a 4 per cent incidence in some racial stock and 6 per cent in others. Gradenigo[18] gave an incidence of 0.2 per cent, while Urbantschitsch[55] estimated 0.19 per cent. Stiles[53] showed that 22 patients out of 38 patients seen had unilateral fistulas. Stannus[52] in his study of African natives gave an incidence of 4.5 per cent—2.08 per cent on the right, 1.69 per cent on the left, and .77 per cent bilateral. More females were affected than males—5.12 per cent females, 3.6 per cent males. He reported primarily the helicine types. One case mentioned and not included in the series was a fistula at the junction of the tragus and lobule. That location is similar to the one described in my second case.

The anatomy and the pathology of the congenital fistula of the auricle will be outlined to include the various locations, but will more specifically treat with the most common one, namely the marginal helicine type. Congdon[8] stated that the skin is not built up about the opening of any of the fistulas. The marginal helicine is situated in line with the direction of the antihelix, a little more often than not, it may be above this line.[50] In no type is the opening large. Its average measurement is 0.75 mm. or less transversely and 1 to 3 mm. vertically. The sac is cutaneous and contains no hairs or glands; the lining, however, may be folded and contain diverticula.[1] Evans stated the sebum is present because of the presence of sebaceous glands and that the direction of the canal is usually downward and forward. Selkirk[50] in his series of cases never demonstrated projection of hair from the sinus openings. The opening tends to be oval, and the secretions may vary from a milky to a caseous substance.

Congdon[8] gave evidence suggesting that few diseased sinuses are bent near their distal and run deeply parallel to the cartilaginous wall of the external auditory canal; therefore, some are not sounded to their entire length. The depth may vary from a mere pit to 20 mm. or more. Canals usually have been demonstrated to end blindly.

The various types of fistulas now will be discussed in regard to different case presentations.

The marginal helicine type, Congdon[8] has calculated, makes up about 90 per cent of all cases presented. It was the first type reported and is the one most often reported up to the present. Some complications of this type of fistula will be discussed. Some case presentations have shown the fistula to communicate with an ulcer abscess or other fistulous openings. Klaber[29] reported cases of this nature; he presented two cases with complications of secondary ulcers 1 cm. ahead and above the tragus. Both were mistreated because the chief emphasis was on treating the ulcer, and the initial marginal helicine sinus and tract were overlooked. In the first case presented in this paper, the extension of the marginal helicine tract manifested itself in the external auditory canal and was mistaken for a furuncle.

Randall[43] cited five cases, three in women and two in men, four of whom had the fistula on the right. One had a persistent aural fistula with abscesses about the tragus. A probe could be passed downward and slightly inward for about an inch without finding any suggestion of connection with the external auditory canal.

Posterior helicine fistulas were first presented by Congdon.[8] He found the sinuses to point inward, the depth being from a mere pit to 4 mm. Selkirk,[50] in his series of cases, noted a few posterior helicine types. He described them as shallow, and said that no problems were obvious.

Helico-lobular types have been rarely reported, and information is not enlightening. Breuer[7] presented a specific, well illustrated case.

Crural fistulas have been specifically reported by two authors, Selkirk[50] 8 cases, and Congdon[8] 11 cases. The fistulas pointed posteriorly and were up to a depth of 6 mm.

Lobular fistulas were reported by Selkirk.[50] The possibility that the ear lobes were pierced at birth, or shortly afterwards, cannot be definitely ruled out. Some races pierce their babies' ear lobes. Ladd and Gross[30] presented one lobular case.

Fistulas of the concha are rather hard to evaluate, as are some of the crural types. A number of patients with openings in the cavum conchae and cymba were noted; but as these are usual sites for blackheads, it is questionable whether or not the openings were true congenital fistulas. I have noted a few pits measuring from 1 mm. to 5 mm. in diameter and about 1 mm. in depth. One patient said that his was a chicken pox mark. Some pits are artificial and may be caused by chronic ear picking, as Aird[1] has suggested.

There is much doubt as to the origin of the posterior auricular fistula in that it is remote from the hillocks. This fistula is usually found at the level of the tragus. Moldenhauer[35] described three such cases. McKenzie reported a fistula behind the lobule on the left side; it was proved to be a redundant external auditory canal and will be discussed

along with other associated anomalies. My second case shows a tract paralleling the left external auditory canal; it was demonstrated by the instillation of lipiodol.

The origin of preauricular fistulas may be primarily infection rather than developmental. They are situated just anterior to the marginal helicine types and may be due to the extension of the marginal tract. These tracts may be repeatedly infected and abscessed. Congdon[8] showed that the sinuses pointed toward the lower end of the ascending limb of the helix. The openings of the preauricular fistula vary considerably in their craniocaudal position, and, therefore, the direction of the sinus tracts is rather unpredictable. The fistulas are always superficial to the deep fascia; therefore, no possible connection with the pharynx or ear can exist. Penich[41] suggested that they may be associated with a subcutaneous epithelial cyst. The sinus and cyst are lined with stratified squamous epithelium and do not demonstrate sweat or sebaceous glands. Preauricular sinuses, therefore, should not be confused with sebaceous cysts, which are usually found posterior to the auricle.

The direction of the sinuses and tracts in adults may be explained as Congdon[8] suggests. The axial arrangement shows how the different types of sinuses have been dragged by a developmental shifting of the skin through which the sinuses open, and the axis of the sinus and of its opening have in consequence aligned themselves in the direction of the traction. The marginal helicine group is affected by the expansion of the cranial vault; therefore the sinus direction is downward, inward and either forward or backward.

The preauricular sinus is associated with the growth of the face. The axis again is inward and seems to be most often directed posteriorly.

The same inward and posterior direction of the posterior helicine sinuses and the axis of the openings may be related to the late growth of the lobule in excess of the rest of the auricle.

The direction of the crural sinus may be governed by the

migration of deeper tissues of the crus from the mandibular toward the hyoid arch early in development, and the skin of the crus to a certain extent may be subjected to the traction in the anterior direction by the growth of the face.

The cicatricial appearance of the skin ahead of the sinus was noted by Heusinger[19] in both the right and left sides. Streeter[54] said that the skin of the cheek below and anterior to the orifice of the preauricular fistula shows an unusual patchy scarring, that it may appear elongated from above downward, and that it may be likened to striae gravidarum. It may be superficial to the deep part of the tract, or it may lie in front of the fundus, with no apparent relation to the tract. Stannus[52] noted scarring over the dilated part of the sinus which may lie in this area. The patchy white scarring may be due to distention of the tissues during an acute phase, such as an abscess. When the swelling subsides, a strial effect may remain; however, these scars may be found with or without preauricular fistulas. Congdon[8] showed cutaneous scars with two preauricular sinuses.

HEREDITARY ASPECTS OF THE PITTED EAR.

Quelprud[42] said that the labile developmental tendency, which is expressed by variation in manifestation of the pitting, seems in most cases to be characteristic of an irrgular or an incomplete dominant. Kindred,[27] reporting four generatings of pitting, noted that the pit may be transmitted by either sex, judging from the manner of its appearance. The marking is neither dominant nor recessive but falls into the intermediate, doubtful class of incomplete dominance. Connon[9] agreed with this manner of inheritance and reported six generations of pitting.

Lewis[31] studied one family through four generations and found that 14 of a total of 33 members had ear pits.

McDonough[32] reported that in identical twins, one had a fistula and one did not. The twin without the fistula had a son who had a fistula. The twin with the pit had two children, neither of whom had pits.

In the series studied by Selkirk,[50] several of the parents of the children were found to have fistulas. Only 17 instances were noted in which more than one child in the family had fistulas, of the margin of the helix. No instances were noted in which more than two children in the same family had fistulas.

Whitney[57] reported three generations of ear pits. A grandfather who had three children had a pit in the left ear. One child had bilateral pits; three of his four children had pits.

Fox[16] reported pits in six members of one family, three of which required surgical correction.

Ballantyne[2] stated that the fistula may have suggested the transmission of an acquired defect produced by ear boring. Ear boring was apparently the practice inflicted upon the infant in very ancient times. The practice continued through many generations. So a pit in an infant may have suggested to a tribe or to mankind generally the practice of ear boring. The traumatism may not have caused the congenital malformation, but in a sense the occasional occurrence of the malformation may have led to the frequent infliction of the traumatism.

Associated anomalies are found, but neither frequently nor with any regularity. Scott and Wooding[49] related other defects associated with the fistula, such as preauricular scars, auricular appendages in the form of tubercles or cartilaginous nodules. Major developmental disturbances other than those of the ear and branchial system are rarely encountered in patients with fistula. Congdon found only two out of 518 patients with abnormalities of the face or the ear.

Mort[37] described a patient with a malformed displaced auricle and with hemiatrophy of the face and paresis of the left side of the face. A dimple above the malformed auricle seemed to be over the location of the internal ear.

Jones and Wen[25] in 1934 presented a case of arrested development of the structures from the mandibular arch; specifically the tragus was missing. Gelston and Sappington[17] encountered a patient with a congenital malformation of the

left auricle and with a facial paralysis and a marginal helicine fistula. The external canal ended blindly one-half centimeter from the entrance. There was a pronounced depression behind the auricle, indicating an abnormality of the middle and probably the internal ears. Roentgenograms, however, showed a seemingly normal internal ear.

Selkirk, in his 172 cases, had one patient with chronic otitis media, one with acute otitis media and two with accessory cartilages of the ear. Two presented gross deformities. One had a microtia, atresia of the external auditory canal and two fistulas—a large one at the margin of the helix, a smaller one representing the ear canal. The other grossly deformed child had a microtia, a normal external auditory canal, a typical marginal fistula and congenital paralysis of the facial muscles. Binnie[4] presented a bilateral atresia case.

Selkirk[50] has said that auricular appendages are much less common than aural fistulas. They are usually found at the level of the spine of the helix or at the upper end of the tragus, below the typical position of the fistula. One patient had three small nodules, the highest one at the site of a marginal fistula, the second one at the spine of the helix, and the third in front of the tragus. Another had a marginal helicine fistula and an accessory cartilage of the same ear with the nodule at a lower level. Another child had a nodule of the tragus on the same side as the fistula. One patient had a tower head and nystagmus. One had incurved little fingers, and two had accessory nipples.

The case report of McKenzie,[33] mentioned with the posterior helicine group, is worth detailed discussion for its help in explaining my second case. McKenzie's patient was a seven-year-old, born with a redundant right auricle, which was partially removed when the child was one year old. An abscess formed repeatedly behind the left auricle. It pointed both into the meatus and behind and below the auricle externally. When it was opened and curettage done, the contents were that of a suppurating dermoid cyst. The redundant auricle was again removed. A sinus drained at the

lower part of the mastoid region, while there was another much smaller opening in the posterior, inferior wall of the external meatus, near the conchal margin. The tympanic membrane was normal.

Surgical operation consisted of a post-auricular incision which exposed what seemed to be an additional external auditory canal. It was removed and found to be a structure about one inch long. The tube was found anterior to the mastoid process and immediately below the normal meatus. The bony floor seemed deficient but otherwise normal. It was easily resected but was bathed in pus. Pathologic study showed sebaceous glands, hairs and mainly tissues and material found in a normal external auditory canal.

Evans[14] has said it is a common fallacy handed down by the scribe from book to book that congenital irregularities of all degrees are more prone to malignant disease than normal tissue.

The treatment for the anomaly has been more or less evolutionary. At first seemingly inadequate means were applied, then were discarded, and now the treatment of choice is complete surgical removal. Congdon,[8] Miller[34] and Bofenkamp[5] and others agreed that the proper treatment is the complete extirpation of the primary and the secondary lesions.

Chemical cautery was used by Randall.[43] Actual cautery was used by Dyer,[12,13] who affected almost a complete cure by employing a hot platinum wire. Roentgen ray, radium, tuberculin and injection of sclerotics have been used.

Dingman[10] pointed out that drainage of secondary fistula will not be cured by use of curettage.

Miller[34] gave indications for surgical intervention in either the acute or chronic infection. Either severe fistulous infection, perichondritis or chondritis of the entire auricle may occur. Drainage may be through the primary fistula or through the secondary opening, as in the cases of Randall[43] and Klaber.[29]

Bofenkamp[5] suggested injection of methylene blue at least

one day prior to surgical operation so the dye which escapes into the tissue adjacent to the tract can be phagocytized and thus leave only the tract stained. Dingman[19] used methylene blue, which was instilled into the tract on three consecutive days before excision. He thought that injection at the time of operation caused adjacent tissue to be stained so that difficulty of operation was increased; he used sharp dissection. Havens[20] used methylene blue at the time of surgery, then used an electric scalpel (radio knife). After opening the sinus and tract the entire region is electrocoagulated sufficiently to destroy the epithelial lining. Healing time was from four to six weeks. There was no conspicuous scarring.

Miller and Moore[34] used methylene blue at the time of surgical operation. An elliptical incision of the skin was made to remove the orifice. Sharp dissection was used with the patient under local or general anesthesia. Postoperative course usually took five to seven days for healing.

REPORTS OF CASES.

Case 1. J.M.W., a white married woman, 19 years old, complained of a recurrent swelling just anterior to the left ear. The lesion which appeared at irregular intervals would open and drain. After drainage, the swelling would subside. Drainage consisted of a substance which was yellowish during most of the episode but which occasionally was bloody toward the end. There had never been very much pain. Three weeks before admission, the patient noticed that a new swelling had appeared just inside of the left ear canal. Drainage was started at this site after the swelling was opened by a doctor. The swelling recurred in a few days after the drainage stopped.

Physical examination showed a well nourished white woman in no distress. Her right ear was normal. The left ear had a small crusted area just ahead of the ascending crus of the helix. There was a second crust opening just inside of the left external auditory canal superiorly. There was no swelling or drainage at the time of examination. A probe was passed from the marginal helicine opening into the external auditory canal.

The patient was admitted to the hospital on October 1, 1951. The surgical procedure was done under local anesthesia. The sinus was injected with methylene blue at the time of surgical operation. A probe was passed into the tract to facilitate the complete excision of the tract. An elliptical incision was used to excise the opening. The incision was carried through the skin covering the tract and to include the opening into the external auditory canal; the tract was removed intact. Sacculations were demonstrated in the length of the sinus. The skin was closed with interrupted silk sutures, and a small rubber drain was left in place (see Fig. 5).

The postoperative course was uneventful. The drain was removed

in two days. Sutures were removed the fourth day (see Fig. 6). The patient was dismissed October 5, 1951, just three days after operation.

The pathologist's report said that the tract was approximately 1.5 cm. by 0.6 cm. by 0.4 cm. Tissue showed extensive chronic infection and formation of granulation tissue. The sinus tract was covered by a squamous epithelium with evaginations in some portions.

Comment: This was a case of marginal helicine fistula which was complicated by the extension of the tract by repeated infection and abscess formation until it actually caused a condition which might be diagnosed as furunculosis of the external auditory canal. The tract was made complete by the incision of the swelling into the external ear canal. A complete, fairly well fibrosed tract was established, and it was totally excised by sharp dissection. Healing was rapid.

Case 2. D.E.C., a white boy one year of age, was first seen because of a recurrent swelling behind his left ear, just back of the ear lobe. Some infection had been present in this area since he was four months old. The swelling had been very obvious on two occasions before he was one year old. The second swelling was incised and drained by his pediatrician. On subsequent episodes of infection, drainage was spontaneous. The referring physician wondered whether the condition could be a mastoiditis with a break through the tip cell of the mastoid. Roentgenogram when the patient was one year old showed infantile mastoids with no definite evidence of cell structure in either mastoid area. There was slightly more sclerosis on the left. Repeat roentgenograms six months later showed the air cells in the right as normal infantile type, while in the left they were aerated and again showed some increase of bone sclerosis. There was no evidence of active inflammatory disease.

Physical examination showed a mild hemiatrophy of the left side of the face. There was an area of discoloration and inflammation posterior to the left ear lobe. This time it felt cystic and superficial (see Fig. 7). There was a congenital fistula of the auricle below the tragus on the left (see Fig. 8). The orifice was 0.3 mm. in diameter and 2 mm. in depth. There was caseous secretion in the tract. The inferior wall of the external auditory canal was pushed upward so as to make the canal smaller than the other canal. This bulging into the canal became plainly obvious at the time of the swelling back of the ear lobe.

The patient was admitted about six months after the first visit, and an operation was performed to remove the fistula and to eradicate the cause of the recurrent swellings. The sinus below the tragus was first injected with methylene blue, and it took stain quite well. The posterior area was injected through the incision site, but no stain could be forced into it. There was no evidence of any connection of the posterior lesion with the infratragal fistula. The posterior area was operated on first. An elliptical incision was used to remove the infected skin, but no tract was found, even though the dissection was carried down to include part of the parotid gland. The anterior sinus below the tragus was completely excised over a probe. No connection was found between the anterior sinus and the posterior infected area. Healing seemed to complete in about two weeks (see Fig. 9).

Pathologic study of the posterior specimen showed that the skin taken from behind the ear was chronically inflamed as from a foreign body. The anterior specimen was compatible with a small fistula. The sinus was lined with a thin squamous epithelium. No inflammation was noted.

The infection posterior to the left ear lobe recurred six weeks after operation. It seemed to be superficial and easily drained. The sac, however, appeared to be deep and was cauterized with 50 per cent silver

nitrate. Suspicion was aroused so that when the area again became swollen the cyst was incised. This time the drainage was a copious, amorphous, acellular substance. Two days later the sac was filled with iodized oil by using a medicine dropper to force it gently into the depths of the sac (see Fig. 10). Roentgenogram showed a tract about 1 inch long parallel to and below the external auditory canal, compatible with a redundant external auditory canal.

Comment: This patient presented a congenital fistula of the auricle in the infratragal area along with confusing associated anomalies. The mild hemiatrophy of the face may account for the difference of the mastoid roentgenograms. The recurring swelling behind the left ear lobe made it necessary to rule out mastoiditis. The redundant tract parallel to and below the external auditory canal was found only after the instillation of iodized oil.

SUMMARY.

Congenital fistulas of the auricle are not rare anomalies, although they frequently are not diagnosed. Improper diagnosis necessarily leads to erroneous treatment. The fistulas are commonly mistaken for sebaceous cysts, abscesses and blackheads.

The origin of the ear and of the ear pits is not completely settled. The hillocks theory of His is most acceptable but does not fulfill the need for an explanation of the development of the whole auricle. The skin fold behind hillocks 4 and 5 is necessary, according to Gradenigo, Schwalbe, and Streeter.

There are several types of fistulas, named according to their location. The most common one is the marginal helicine; others are posterior helicine crural, lobular, helicolobular, postauricular and preauricular. An infratragal fistula is described in this paper.

Heredity plays a part in the distribution of the ear pits. It is thought to be characteristic in most cases of an irregular or an incomplete dominant.

Treatment has been varied, from chemical cautery, repeated incisions to complete surgical removal, which now is the treatment of choice.

The first case presented was primarily a marginal helicine fistula, which was complicated by the extension of the tract into the external auditory canal by recurrent infection and

finally was made into a common tract by incision of the swelling into the upper part of the external ear canal. Complete excision affected a direct cure.

The second case was an infratragal auris fistula of the left ear. Associated anomalies were mild hemiatrophy of the face on the left and a redundant tract demonstrated by iodized oil instillation. The tract paralleled the external auditory canal and seemed to extend as far as the canal.

BIBLIOGRAPHY.

1. Aird, I.: Ear Pit (Congenital Aural and Preauricular Fistula). *Edinburgh Med. Jour.*, 53:498, Sept., 1946.

2. Ballantyne, J. W.: "Manual of Antenatal Pathology and Hygiene," Vol. 2, p. 442. Green, Edinburgh, 1904.

3. Becker, S. W., and Brunschwig, A.: Sinus Preauricularis (Fistula Preauricularis Congenita). *Am. Jour. Surg.*, 24:174, April, 1934.

4. Binnie, J. F.: Remarks on Some Cases of Deformity of the External Ear. *Ann. Surg.*, 24:206, July-Dec., 1896.

5. Bofenkamp, Benjamin: Congenital Auris Fistula. *Minnesota Med.*, 34:329, April, 1951.

6. Brash, J. C.; Jamieson, E. B., and Cunningham, Daniel, J.: "Textbook of Anatomy," 7th Ed. Oxford Univ. Press, New York, 1937.

7. Breuer, Fritz: Congenital Fistula of the Auricle. *Deutsche Ztschr. f. Chir.*, 202:278, May, 1927.

8. Congdon, E. D.; Rowhanavongse, S., and Varmisara, P.: Human Congenital Auricular and Juxta-auricular Fossae and Sinuses and Scars (including the so-called aural and auricular fistulae) and the Bearing of Their Anatomy upon the Theories of Their Genesis. *Am. Jour. Anat.*, 51:439, Nov., 1932.

9. Connon, F. E.: Inheritance of Ear Pits in Six Generations of a Family. *Jour. Hered.*, 32:413, Nov., 1941.

10. Dingham, R. O.: Congenital Sinus. *Arch. Otolaryngol.*, 29:982, June, 1939.

11. Doran, A.: Case of Congenital Auricular Sinus. *Trans. Obst. Soc.*, London, 33:199, June, 1891.

12. Dyer, E.: Fistula of the Antitragus. *Med. News*, 46:24, Jan. 3, 1885.

13. Dyer, E.: Fistula Auris. *Trans. Am. Otol. Soc.*, 3:468, 1885.

14. Evans, Howell: Auricular and Periauricular Dermoids, Fistulae and Tumors of Congenital Origin. *Brit. Jour. Child. Dis.*, 7:490, 1910.

15. Federici, F.: The Embryological Interpretation of Congenital Preauricular Fistulae. *Acta. Oto-Larygol.*, 14:532, 1930.

16. Fox, M. S.: Congenital Aural Fistula. *Arch. Otolaryngol.*, 35:431, March, 1942.

17. Gelston, C. F., and Sappington, E.: Congenital Malformation of

the Auricle Associated with Paralysis of the Facial Nerve. *Arch. Ped'at.*, 49:730, Nov., 1932.

18. Gradenigo, G.: Die Formentwickelung der Ohrmuschel, mit Ruchsicht auf die Morphologie und Teratologie Derselben. *Centralbl. f. d. Med. Wissensch.*, 26:82, 1888.

19. Heusinger, Rath: Hals-keiman-fistein von noch Nicht Beobachteter. *Virchow's Arch. f. Path. Anat.*, 29:358, 1864.

20. Havens, F. Z.: Preauricular Fistula, Congenital Branchiogenic Sinus and Treat. *Arch. Otolaryngol.*, 29:985, June, 1939.

21. His, W.: "Aufstellung von Entwickelungsnormen z. Weiter Monat. Anatomie Menschlicher Embryonen," Pt. 2, 60. F. C. W. Vogel, Leipzig, 1882.

22. His, W.: Die Formentwickelung der Menschlichen Ohrmuschel, Compt. Rend. *Cong. Internat. d'Otol.*, 3:149, 1885.

23. His, W.: "Die Formentwickelung des Ausseren Ohres. Anatomie Menschlicher Embryonen," pt. 3, 211, 1886.

24. Hyndman, O. R., and Light, G.: The Branchial Apparatus. *Arch. Surg.*, 19:410, Sept., 1929.

25. Jones, F. W., and Wen, I. C.: Development of External Ear. *Jour. Anat.*, 68:525, July, 1934.

26. Keith, A.: Congenital Malformation of Palate, Face and Neck. *Brit. Med. Jour.*, 2:438, 1909.

27. Kindred, J. E.: Inheritance of a Pit in the Skin of the Left Ear. *Jour. Hered.*, 12:366, 1921.

28. Kipp, C. J.: On Branchial Fistula of the External Ear. *Trans. Am. Otol. Soc.*, 2:402, July 21, 1880.

29. Klaber, R.: Preauricular Fistulá; Two Cases. *Proc. Roy. Soc. Med.*, 28:1553, Oct., 1935.

30. Ladd, W. E., and Gross, R. E.: Congenital Branchiogenic Anomalies; A report of 82 Cases. *Am. Jour. Surg.*, 39:234, 1938.

31. Lewis, F. P.: A Peculiar Inherited Ear Mark; Fistula Auris Congenita. *Jour. Ophth., Otol. and Laryngol.*, 3:14, 1891.

32. McDonough, E. S.: Inheritance of Ear Pits (fistula auris congenita) with Special Reference to Twins. *Jour. Hered.*, 32:169, May, 1941.

33. McKenzie, D.: Congenital Redundant External Meatus. *Brit. Jour. Child. Dis.*, 17:30, Jan.-March, 1920.

34. Miller, T. B., and Moore, P. M., Jr.: Fistula Auris Congenita. *Arch. Otolaryngol.*, 51:245, Feb., 1950.

35. Moldenhauer, W.: Die Entwicklung des Mittleren und des Ausseren Ohres. *Morph. Jahrb.*, 3:106, 1877.

36. Montgomery, M. L.: Congenital Auricular Fistula. Report of Three Cases in the Same Family. *S. Clin. N. Amer.*, 11:141, 1931.

37. Mort, Spencer: Development of the Auricle on Second Branchial Cleft. *Lancet*, 207:322, Aug. 16, 1924.

38. Onodi, S.: Ueber Krongenitale Ohrfisteln. *Arch f. Ohren, Nasen u Kehlkopfh.*, 102:128, April 26, 1918.

39. Paget, J.: Cases of Branchial Fistulae in the External Ears. *Med. Chir. Trans. London*, 43:41, 1878.

40. PATTEN, BRADLEY M.: "Human Embryology," p. 425. The Blakiston Co., Phila. and Toronto, 1946.

41. PENICH, R. M., JR.: Preauricular Sinuses, Diagnosis and Treatment. *South. Med. Jour.*, 38:103, Feb., 1945.

42. QUELPRUD, J.: Ear Pits and Its Inheritance, Fistula Auris Congenita, Described in 1864, Still Genetical and Embryological Puzzle. *Jour. Hered.*, 36:379, Sept., 1940.

43. RANDALL, B. A.: Some Cases of Supernumerary Auricle and Congenital Aural Fistula. *Trans. Am. Otol. Soc.*, 4:485, 1889.

44. SCHWABACH, D.: On Branchial Fistula at the External Ear. *Arch. Otolaryngol.*, 8:246, 1879.

45. SCHWALBE, G.: Beitrage zur Anthropologie des Ohres. *Internat. Beitr. z wiss. Medicin Festschr. R. Virchow*, 1:95, 1891.

46. SCHWALBE, G.: Ueber Auricularhocker bei Reptilien; ein Beitrag. zur Phylogenie des Ausseren Ohres. *Anat. Anz.*, 6:43, 1891.

47. SCHWALBE, G.: Beitrage zur Kenntnis des Ausseren Ohres der Primaten. *Ztschr. f. Morphol. u. Anthropol.*, 19:545, 1897.

48. SCHWALBE, G.: "Das Auserre Ohr in Handbuch ber Anatomie des Menschen," Pt. 1, 5:113. G. Fischer, Jena, 1897.

49. SCOTT, ROLAND B., and WOODING, CLINTON H., JR.: Congenital Aural Fistula. *Jour. Pediat.*, 36:784, June, 1950.

50. SELKIRK, T. K.: Fistula Auris Congenita. *Am. Jour. Dis. Child.*, 49:431, 1935.

51. STAMMERS, F. A. R.: Preauricular Fistulae. *Brit. Jour. Surg.*, 14:359, 1926.

52. STANNUS, H. S.: Congenital Anomalies in a Native African Race. *Biometrika*, 10:1, 1914.

53. STILES, K. A.: Inheritance of Pitted Ear. *Jour. Hered.*, 36:53, Feb., 1945.

54. STREETER, G. L.: Development of the Auricle in the Human Embryo. *Contrib. Embryol.*, 14:111, 1922.

55. URBANTSCHITSSCH, V.: Concerning the So-Called Fistula Auris Congenita. *Edinburgh Med. Jour.*, 23:690, 1878.

56. VIRCHOW, R.: Ueber Missbildungen Ohr und in Bereiche des Ersten Keimenbogens. *Virchow's Arch. f. Path. Anat.*, 30:221, 1864.

57. WHITNEY, D. D.: Three Generations of Ear Pits. *Jour. Hered.*, 30:323, Aug., 1939.

THE EFFECT OF HEAD-MOVEMENT ON POSITIONAL NYSTAGMUS—ELECTRO-NYSTAGMOGRAPHY WITH AN ELECTRIC DRIVEN POSTURE-TABLE.*†‡

G. ASCHAN, M.D.;
M. BERGSTEDT, M.L.,
B. DRETTNER, M.L.,
C. O. NYLÉN, M.D.,
and
J. STAHLE, M.L.,
Uppsala, Sweden

In the examination of patients with positional nystagmus the following questions arise:

1. Is the releasing factor, the *actual position* of the head?

2. Is the *change in position* of the head of significance in precipitating nystagmus?

This problem has a clinical aspect: how to perform the posture test in clinical practice, on a very slow turning posture-table, or on a couch with fairly rapid or brisk movements of the patient's head?

Bárány, Borries, and Nylén (1921-1924) discussed these points in detail. The problem has recently again become active as a result of investigations by Williams, Lindsay, Dix and Hallpike, McNally, Cawthorne, Frenzel, and Stenger (1947-1955). None of all these investigators has, however, given a definite answer to the raised problem, or a determining proof of the best practical way to do the posture-test.

In 1956 Stenger declared that in examinations of "Lager-

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†From the Oto-Laryngological Department, Royal Academic Hospital, Uppsala, Sweden. Head: Professor C. O. Nylén, M.D.

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ungsnystagmus" the ideal way to produce a sub-threshold rotation effect, although impossible to perform at present, would be a special electrically driven chair. In 1956, however, Aschan, Drettner, and Nylén published a preliminary report on exactly this kind of device.

In order to establish the importance of the various factors, we have devised a posture-table resembling earlier models (Quix, Grahe, Fromm and Nylén, 1926-1935), in which the patient's head can be arranged in any position, but with accurate control of the turning. After initial subliminal acceleration it can be rotated by means of motors with constant speed, according to Nylén's ideas (1950, 1953).

To record nystagmus, we have employed the modern method of nystagmography (registration of the displacements of the cornea-retina potential, due to movements of the eyes, according to Scott-Meyer), in which the patient's eyes are kept closed. For further details of the method we refer to Aschan, Bergstedt, Stahle (1955-56). By this means an objective record of nystagmus is obtained, all visual stimuli being eliminated.

In other investigations a graphical system has been employed (Aschan, Bergstedt, Goldberg, Laurell, 1955-56), which enables one to distinguish the direction and the intensity of nystagmus, even though the experiment continues over a long time and includes an extensive length of nystagmus records. The intensity of nystagmus is best shown as the eye-speed in the slow phase of nystagmus.

THE ELECTRIC POSTURE-TABLE.

The new table is illustrated in Fig. 1. It consists of two independently-moving frames, each driven by a ¼-h.p. direct-current motor, the number of revolutions per minute being regulated by rheostats. The motors are geared at fixed ratio to the axle-shafts of the table. We have used an initial acceleration of 0.05 - 0.1° per second, later rotating the patient at a constant rate. The speed of rotation can be regulated from 0-3° per second.

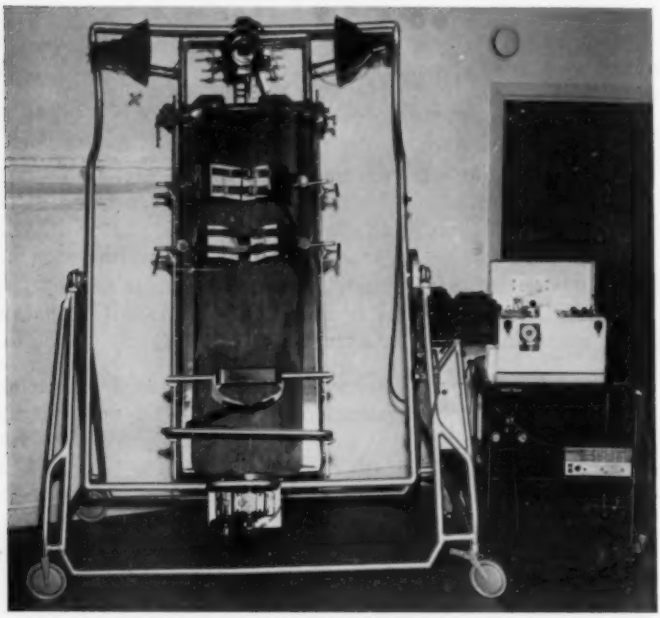

Fig. 1. Electric posture-table. The inner frame is driven by the lower motor, the outer frame by the motor to the right.

To date we have placed the patients horizontally on the posture-table and rotated them about a cranio-caudal axis. Fig. 2 shows the table with the patient lying in a practically left-lateral position, corresponding to 280°.

NYSTAGMOGRAPHY.

Fig. 3 shows several nystagmus records of the same patient, a man of 25 years, who complained about positional vertigo. All the records refer to the supine position. The top tracing (1) shows nystagmus beating to the left with an intensity of about 2° per second with the patient lying in the supine position in the electrically driven posture table. When he was turned around the craniocaudal axis, with a constant speed of 1° per second, the left-beating nystagmus changed

very little. The second record from the top represents the nystagmus after turning 360° in 360 seconds. The intensity is about the same as before starting the experiment. The third curve shows the nystagmus in the supine position after a quick turn from the right lateral into the supine position. The result was a left-beating nystagmus with high intensity

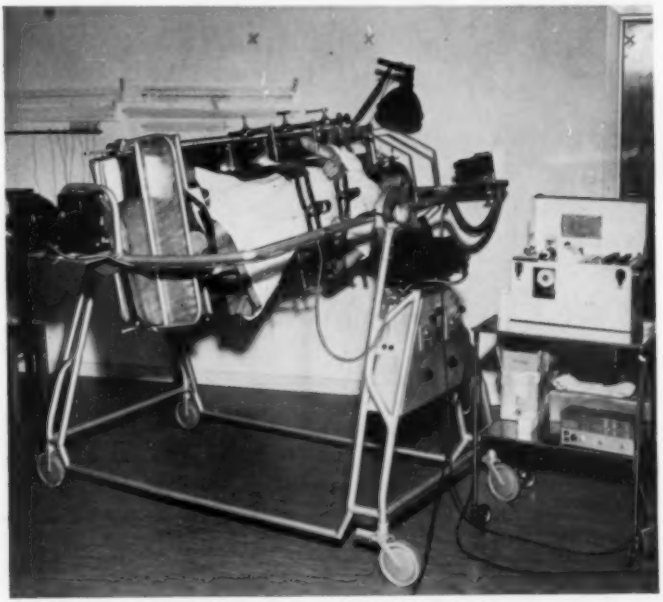

Fig. 2. Posture-table, with the subject rotating about a craniocaudal axis in the horizontal plane, position 250°. Controls situated below the right hand motor. Nystagmograph on the table to the right, with leads in position for recording.

up to a maximum of 40° per second, gradually diminishing in intensity during the following minutes. Curve No. 4 is a record of nystagmus two minutes later when the patient had remained in the supine position. In curve No. 5 the same experiment was performed as in No. 3. This time, at first a few beats of moderate strength to the left were recorded during the turn. Still in the supine position the nystagmus

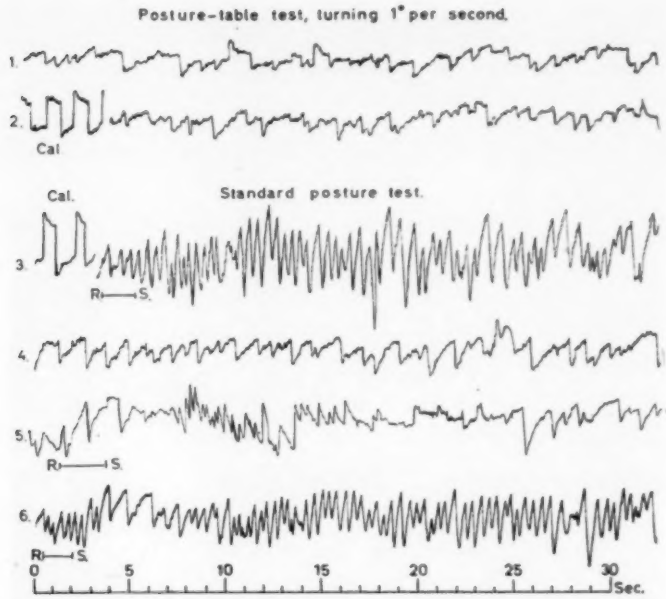

Fig. 3. Nystagmus tracings in one and the same patient at the same examination. The electrodes arranged horizontally. A deviation upwards in the record corresponds to an eye movement to the right, downwards to the left. Cal. means calibration for 10° eye movement in the plane of the electrodes. Tracings 1 and 2 show a left-beating nystagmus, with the same intensity before 1 and after 2, a 360° turn at constant speed in the posture-table. R and S indicates the time during which the patient was turned from right lateral to supine position. In tracings 3 and 6 this maneuver results in an intense left-beating nystagmus. Curve 4 refers to the same test as curve 3, but shows that two minutes later the nystagmus still has a higher intensity than compared to 1 and 2. Record No. 5 shows a right-beating transitional nystagmus later changing to left-beating after the same maneuver as in tracings 3 and 6. All the tracings refer to the supine position.

changed direction to an intense right-beating one, lasting over a period of 10 seconds, and then followed by left-beating nystagmus. In the next curve (No. 6) the same maneuver was performed again, but this time only a left-beating nystagmus with very high intensity was recorded.

In the above-mentioned patient, where the nystagmus recorded referred to only one and the same position the nystagmus must be termed a positional direction-changing and

transitional nystagmus, Type III, according to Nylén's classification system.

These records in Fig. 3, and those from the same patient in other positions, can be reproduced in a graph, including the direction and intensity of nystagmus and the position to which the records correspond.

In Fig. 4 the results from three typical cases are brought together, the bottom graph corresponding to Fig. 3 with all

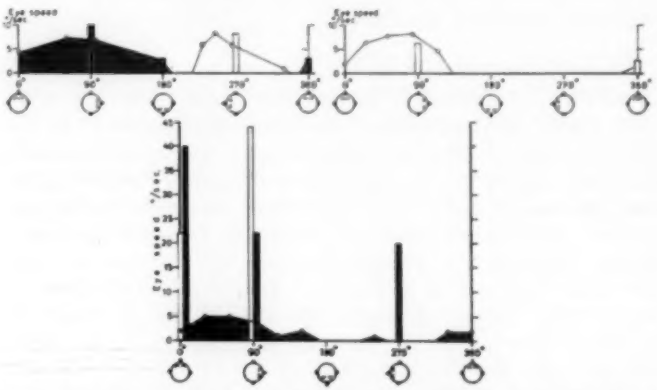

Fig. 4. Qualitative and quantitative comparisons between posture-table tests and standard posture tests (piles). Three patients, one with positional nystagmus Type 1 (top left), Type 2 (top right) and Type 3 (bottom). Along the abscissa is shown the position of the head along the ordinate, the intensity of nystagmus given as the eye-speed in the slow nystagmus-phase. White areas indicate right-beating, and black fields left-beating nystagmus. In the persistent positional nystagmus Type 1 and 2 the results of the two tests correspond fairly well in both direction and intensity. In Type 3 there is no such correlation.

the records from the supine position. Along the abscissa are shown the positions of the patient's head, 0° means supine, 90° right lateral, 270° left lateral position, etc. Black area indicates a left-beating, and white a right-beating nystagmus. The ordinate gives the intensity of nystagmus as the eye-speed in the slow phase of nystagmus in degrees per second. The continuous area indicates the results when the patient is turned very slowly at a constant rate in the posture table. The bottom graph shows in the patient demonstrated earlier,

a left-beating nystagmus with an intensity never exceeding 5 to 6° per second, and refers to the slow-turning posture table test. The piles, on the other hand, corresponding to supine (0°), right lateral (90°) and left lateral (270°) positions indicate the maximum eye-speed recorded when the patient was briskly brought into the different positions. The result is a nystagmus beating either to the right or to the left, and of a very high intensity compared to the test in the posture table. When repeating the posture test, different types of nystagmus were recorded, indicated by two piles at the same position. The intensity of nystagmus refers to the maximum eye-speed obtained.

The two other graphs in Fig. 4 refer to two patients with a positional nystagmus Type I, direction changing, top graph to the right; another with a positional nystagmus Type II—direction fixed. In these two patients the nystagmus was persistent, which means that it had the same intensity when observed over a period of at least 60 seconds in the same position. In these two patients there was, however, a striking parallel between the results obtained in the posture table and those obtained in a standard positional test, when the patient was quickly brought into the different positions. Repeated tests either in the posture table or on the couch gave the same results in both cases.

The results demonstrated in these three patients (Figs. 3 and 4), representing the three main types of positional nystagmus are typical for a larger material and can be summed up as follows: when dealing with *persistent forms of positional nystagmus the position of the head is the determining factor for releasing nystagmus. For transitory and transitional forms of positional nystagmus the movement of the head into the new position also plays an important part, often the dominating one in the releasing mechanism.* It is a common finding that in a patient an intense transitory positional nystagmus Type III observed after brisk head-movement can not be reproduced when turning slowly in the posture table.

The practical clinical consequences of this investigation is

that the most suitable way to perform the examination for positional nystagmus is the fairly rapid turning of the patient in the different positions. When turning slowly into different positions transitory forms of positional nystagmus might be overlooked. *This posture test corresponds on the whole to the old fashioned test on a couch.*

The investigation also stresses the value of recording nystagmus, thus making it possible to compare quantitatively and qualitatively nystagmus released under different test conditions.

SUMMARY.

The use of a posture table should facilitate the differentiation of the significance of head-movement and head-position in the release of positional nystagmus. An electrically driven posture table is described. With this it is possible to rotate the patient at a constant rate following initial subliminal acceleration.

Experiments in an electric posture table, on patients with persistent and transitory positional forms of nystagmus, are described. Nystagmus was recorded instrumentally with the patient's eyes closed. This precaution eliminates the source of error due to visual stimuli. In addition, nystagmus can be assessed both qualitatively and quantitatively, and comparisons can be made between records obtained under different test conditions.

The investigation indicates that in persistent forms of positional nystagmus the position of the head is the determining factor. In transitory and transitional forms, on the other hand, movements of the head also play a significant part in the releasing mechanism.

To avoid overlooking cases with transitory forms of positional nystagmus, the most suitable way to perform the standard posture-test, therefore, is to let the patient perform a fairly rapid turning from one position of the head to another. It corresponds on the whole to the old fashion of testing on a couch.

REFERENCES.

ASCHAN, G.: The Mechanism of the Cupula Ampullaris in Man. *Acta Soc. Med. Upsaliensis*, 60:77, 1955.

ASCHAN, G.: The Caloric Test. *Acta Soc. Med. Upsaliensis*, 60:99, 1955.

ASCHAN, G., and BERGSTEDT, M.: Non-vestibular Nystagmus; a Nystagmographic Investigation. *Acta Soc. Med. Upsaliensis*, 60:1, 1955.

ASCHAN, G.; BERGSTEDT, M.; GOLDBERG, L., and LAURELL, L.: Alkohollägenystagmus. *Svensk Otolaryngologisk Forenings Forhandlingar*, 1955.

ASCHAN, G.; BERGSTEDT, M.; GOLDBERG, L., and LAURELL, L.: Positional Nystagmus in Man During and After Alcohol Intoxication. *Quart. Jour. Studies on Alcohol*, 17:381, 1956.

ASCHAN, G.; BERGSTEDT, M., and STAHLE, J.: Nystagmography. *Acta Oto-laryngol.*, Suppl. 129, 1956.

ASCHAN, G.; DRETTNER, B., and NYLÉN, C. O.: Rörelsemomentets betydelse vid lägenystagmus. Registrering av nystagmus i ett elektriskt drivet lägebord. *Svensk Otolaryngol. Forenings Forhandlingar*, häfte 3, 1956. Swedish; English summary.

ASCHAN, G.; DRETTNER, B., and NYLÉN, C. O.: Die Bedeutung des Bewegungsmomentes bei Lagenystagmus. Nystagmusregistrierung in einem elektrisch getriebenen Lagetisch. *Mschr. f. Ohrenheilk*, 91:151, 1957.

BARANY, R.: Diagnose v. Krankheitserscheinungen im Bereiche des Otolithenapparates. *Acta Oto-laryngol.*, 2:434, 1921.

BORRIES, G. V. TH.: Klin. Untersuch. über die durch Kopfbeweg. und Kopfstell. ausgelöst. Nystagmusanfälle. *Mschr. Ohrenheilk.*, 57:644, 1923.

CAWTHORNE, T.: Positional Nystagmus. *Ann. Otol., Rhinol. and Laryngol.*, 63:481, 1954.

DIX, M. R., and HALLPIKE, C. S.: The Pathology, Symptomatology and Diagnosis of Certain Common Disorders of the Vestibular System. *Proceed. Royal Soc. of Med.*, 45:341, 1952.

FISCHER, J. J.: *The Labyrinth*. New York-London, 1956.

FRENZEL, H.: Spontan- und Provokations-nystagmus als Krankheitssymptom. Berlin, 1955.

FROMM, B., and NYLÉN, C. O.: Cinematogr. film of posit. nyst. *Acta Oto-Laryngol.*, 22:370, 1935.

GRAHE, K.: Die Vertikalempf. auf d. "Vestibular-Tische". *Acta Oto-Laryngol.*, 11:159, 1927.

LINDSAY, J. R.: Postural Vertigo and Positional Nystagmus. *Ann. Otol., Rhinol. and Laryngol.*, 60:1134, 1951.

MCNALLY, W. J.: Some Remarks About Dizziness—Its Diagnostic Significance and Treatment. *Ann. Otol., Rhinol. and Laryngol.*, 62:607, 1953.

NYLÉN, C. O.: Some Cases of Ocular Nystagmus Due to Certain Positions of the Head. *Acta Oto-Laryngol.*, 6:106, 1924.

NYLÉN, C. O.: Positional Nystagmus. *Jour. Laryngol-Otol.*, 64:295, 1950.

NYLÉN, C. O.: Posture Test. *Acta Oto-Laryngol.*, Suppl. 109:125, 1953.

QUIX, F. H.: Ned. K.N.O. Veren., Maj 1926, Cit. after Jongkees, L.B.W., Fortschr. *Hals-Nas.-Ohrenhk.*, 1:43, 1953.

STENGER, H. H.: Über Lagerungsnystagmus unter besonderer Berücksichtigung des gegenläufigen transitorischen Provokations-Nystagmus nach Lagewechsel in der Sagittalebene. *Arch. Ohr., Nasen, Heilk.*, 168:220, 1955.

WILLIAMS, H. L.: The Present Status of the Diagn. and Treatm. of Endolymphatic Hydrops. *Ann. Otol., Rhinol. and Laryngol.*, 56:614, 1947.

CORRECTION.

In the paper "A Study of 500 Stapes Mobilizations," by Victor Goodhill, M.D., and A. L. Holcomb, E.E., published in the July issue of THE LARYNGOSCOPE, the running heads should read "500 Mobilizations".

In Figure 8, in the blank space at the top should appear the words "Operative Failures and Early Closures".

On page 623 under caption 5: Antibiotics, third line the word "Mycostatin" should read "Nystatin".

CUPULOMETRY.*†

J. J. GROEN, M.PHIL.,

Utrecht, Netherlands.

INTRODUCTION.

The examination of the vestibular organs as initiated by Bárány, Sr., and others, was founded on a wrong interpretation of the function of the semicircular canal system. In those days (1905) the cupula would not reach farther than half-way to the roof of the ampulla (see Fig. 1-A). It is now known that this was due to a shrinking process caused by the chemical treatment and the preparation necessary for microscopic analysis. A cupula thus deformed, together with the endolymph, would provide a mechanism (Mach 1875)[9] totally different from the cupula which reaches the roof of the ampulla and seals it (see Fig. 1-B). A shrunken cupula is totally passive with regard to the endolymph: an impulse will cause the endolymph to travel a short distance. During this displacement, the cupula will bend outward as a reed in the wind. The moment the endolymph stops, the cupula raises its head and nothing of prolonged duration could be expected afterwards. Only during the displacement could any activity from the cupula-crista sensory structure be noticed, but it was known that there were after-phenomena, as, for instance, nystagmus and sensation of long duration. These could be explained only by supposing that a central process was responsible. Mechanically, nothing would prevent its giving two impulses of even and opposite sign in rapid succession, because the endolymph would stop so quickly that the responses to each of the stimuli would not be affected by each other.

The evaluation of the central processes started by these

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†State University, Utrecht, Netherlands, Department of Otorhinolaryngology. Director: Prof. Dr. A. A. J. van Egmond.

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two counteracting stimuli was another matter, which was treated only in vague terms. Viewed from this standpoint, it was permissible for Bárány to introduce his technique, which is still in use: the test subject is seated on a rotating chair, head inclined 30° forward. He is submitted to 10 revolutions in 20 seconds, beginning with a quick start, followed by a sudden stop. The duration of the nystagmus is taken as a measure of the sensitivity of the semicircular system. On the average, 22 seconds is needed for the eyes to be at rest again; yet there is a large amount of scatter-

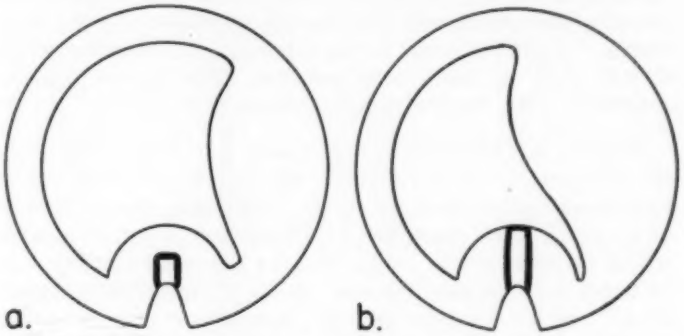

Fig. 1-A. Cupula, shrunken by chemical treatment. The endolymph will pass over it, causing it to bend sideward only during the flow. Short duration of cupula-crista activity.

Fig. 1-B. Cupula in normal condition; bent sideward by an endolymph displacement, but taking over as soon as the endolymph stops. The normal cupula will push the endolymph slowly back through the narrow canal. Long duration of cupula-crista activity.

ing, which has no immediate connection with difference in sensitivity between the test subjects.

Steinhausen,[11] 1932, discovered that the cupula in the living animal reaches to the roof of the ampulla, acting like a hermetically sealing swinging door with spring-like properties. This produces a totally different picture, and consequently the technique of examination must be modified according to the mechanics of the real system; van Egmond et al.,[1] 1943. Suppose we repeat the experiment mentioned above: the test subject is quickly started to rotate

with a constant speed. The endolymph, by its inertia, travels the same distance (almost) as in the former case; but now the difference is noticed: the cupula is jerked outward and starts to come back *slowly*, because it has to push the endolymph through the narrow canal. In the end, which may be as much as one minute (or even more) the cupula will have regained its equilibrium position; thus, there is a strict coupling of the cupula and endolymph. Only during the first stage (the jerk) is the cupula passive. As soon as the endolymph stops, because of the friction, the cupula takes over and drives it slowly back. If we allow the cupula only 20 seconds to return to zero position, the second impulse, the stopping movement, will find the cupula still on its way backwards. It will be jerked to the opposite direction from which it will start to reach zero position. The second stimulus interferes with the declining response to the first.

Suppose we have two test subjects, who differ only as to the stiffness of their cupulae: one cupula, for instance, is two times stiffer than the other. Assuming the friction to be normal in both cases, decay constants of 10 and 20 seconds would be appropriate. Subjected to the same Bárány test, 10 turns in 20 seconds, the test subject with the stiffer cupula would require 21 seconds after nystagmus and the other one, 36 seconds.

There is of course a difference in duration, but this is not proportional. If one wishes to determine the properties of a canal system accurately, this system should be left alone until the cupula-deviation has subsided, which takes even more time than is necessary for crossing the threshold (see Fig. 2). This is done in "Cupulometry," 1942; here the mechanics of the system have been taken into account. Whatever procedure of examination is used, it must have its basis in the mechanism, otherwise the interpretation of the measurements is doubtful.

CUPULOMETRY.

During the last fifteen years "Cupulometry" has been developed and its usefulness has been demonstrated beyond

any doubt (Hulk 1949).[7] Based as it is on purely mechanical laws applied to a biological system, its mathematical simplicity on the one hand and its physiological complications on the other, should have found a happy symbiosis after so many years. Is this really true? Several times the mechanical aspect was obscured by inexplicable phenomena; it

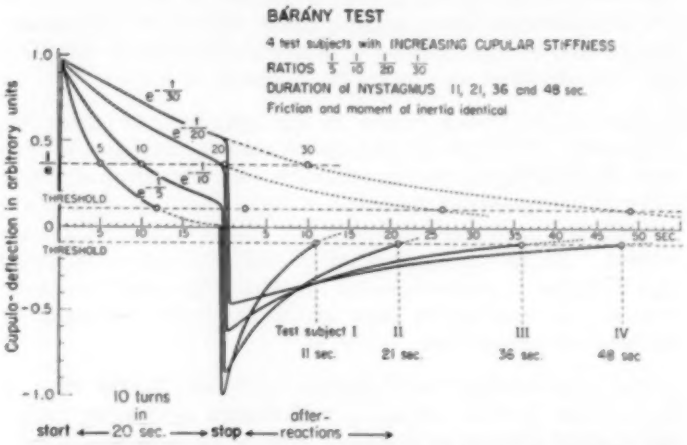

Fig. 2. Four test subjects having similar friction and moment of inertia of the endolymph, but differing as to the stiffness of their cupulae: stiffness ratio 1/5:1/10:1/20:1/30. In this case, the weakest cupula (1/30) will give the longest after-reactions (sensation and nystagmus). If these four test subjects are submitted to the same turning test, the initial cupula deflections will be the same, but the return to equilibrium will be different, depending upon the stiffness. They will need 5, 10, 20 and 30 seconds respectively to pass the 1/e value and 12, 23, 47 and 70 seconds to cross the (identical) threshold. The Bárány test provides the after-reaction of respectively 11, 21, 36 and 48 seconds duration. In the whole range from stiff to infinitely weak cupulae there is a minimum stiffness beyond which the after-reaction to the Bárány test will be shorter the weaker the cupula is. So there are two stiffness values corresponding to each duration of a Bárány test.

seemed that the mathematical laws, although quite true in themselves, could not be applied to a semicircular canal system. This conflict has led to a new approach to the problem of central inhibition. The mechanical point of view was saved by a narrow margin, and our concepts on the evaluation by the central nervous system of the signals arriving there from the peripheral organs have been broadened

and deepened. It is time to reconsider the basis of cupulometry to ascertain the validity of the mechanical foundation, so that we can build on it when we turn to the complications encountered in vestibular disorders.

The Mechanical Basis:

A semicircular canal system, together with the utricle, forms an endoylmph ring with a moment of inertia Θ, which can be calculated from the anatomical data on the dimensions of the canal. The friction π of the endolymph in the canal can be calculated also, since its viscosity is known.

The cupula, fitting closely in the ampulla, has spring-like properties. It keeps the endolymph ring in place but is deviated from its equilibrium position by any angular acceleration having components in the plane of that canal. Its stiffness Δ cannot be calculated beforehand. Some experiments are needed for the determination of that constant. It would also be better to determine experimentally the other constants, because the anatomical data are likely to be distorted by histological procedures.

The semicircular canal system with an inertia ring, a spring and friction, forms a well known mechanism called the damped torsion pendulum.

To learn the properties of such a pendulum it should be oscillated and its period measured. Unfortunately, this cannot be done in the case of the cupula endolymph system because the damping is so high that it will creep back to its equilibrium position.

As the system will not show any free oscillations, we submit it to forced oscillations, and by observing the phase difference between force and cupula as a function of the frequency of the oscillation we may learn its latent natural period. If any oscillatory system is forced to vibrate in its natural period, there always will be a phase difference of 90° between the driving force and the resonating system, whatever the damping may be. In the case of the cupula-endolymph system this could be used in the following manner: the test subject sits on a torsion-swing, which will oscillate

in a horizontal plane; the swing is started, and the test subject is asked to indicate the moment he thinks he reaches his "subjective" turning point. It seems that the average human test subject in favorable conditions will lead with his sensations of swinging, if the period of the swing is greater than 6 seconds. Below this value, his turning points will lag behind those of the swing. By approaching from either side, the frequency at which his sensation of swinging will exactly coincide with the real one can be determined; coincidence means 90° phase lag. The moment the swing

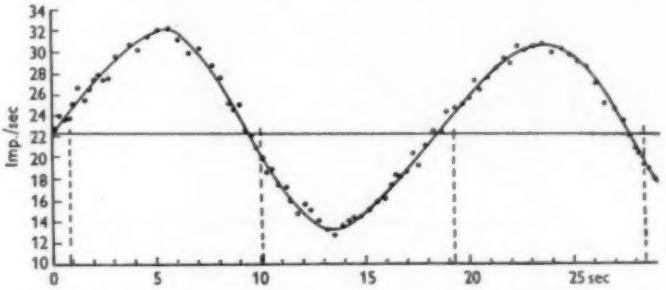

Fig. 3. The frequency of nerve impulses as a function of time during a sinusoidal movement. T=18.4 sec; $\omega=2\pi/T=0.34$ sec^{-1}; A=54°; $\omega A=18.5°$/sec. Total response=9.2 impulses/sec; sensitivity=0.50 impulses/degree; phase lead=13.5±2°. The turning points of the torsion swing are marked by the broken vertical lines.

reaches its outer position, thus coming to a standstill during an infinitesimal time interval, the cupula, lagging 90° behind, will pass zero position, thus also causing a sensation of rest. In this way the average natural period of a series of favorable subjects was established to be about 6.3 seconds (van Egmond, l.c.)[2]. It has always been difficult to explain why the majority of so-called normal people will not respond adequately to this swing test: they either tend to lead permanently, or to give vague answers.

If it were possible to determine the phase difference quantitatively, all the properties of the cupula-endolymph pendulum system could be calculated.

This has been done with a test animal, the ray (Groen et al., 1952)[5] (see Figs. 3 and 4). Up to this time this was impossible with human test subjects; so, we had to rely on another test procedure to obtain the missing data. This can be realized in the so-called "clinical turning test". The test subject is seated on a revolving chair. He is turned around with a constant angular velocity until he feels no rotation; this could have been obtained also by a subliminal acceleration.

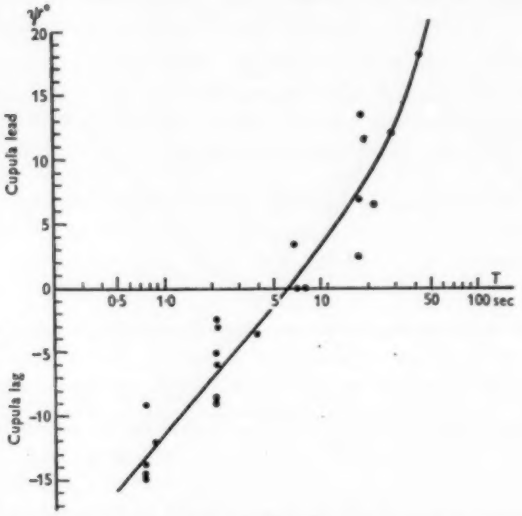

Fig. 4. Phase difference (ψ) of the impulse frequency (deviation of the cupula) in relation to the sinusoidal stimulus as a function of the logarithm of the 'swing' time (T).

As soon as the test subject is in perfect equilibrium, he is suddenly stopped (within 2 seconds). The impulse momentum of the endolymph ring pushes the cupula into a deviated position; the friction in the canal determines how far the endolymph will be displaced. Afterwards the cupula will try to regain its equilibrium by pushing back the endolymph ring. This takes considerable time, and during this period of restoration the test subject will have a declining sensation

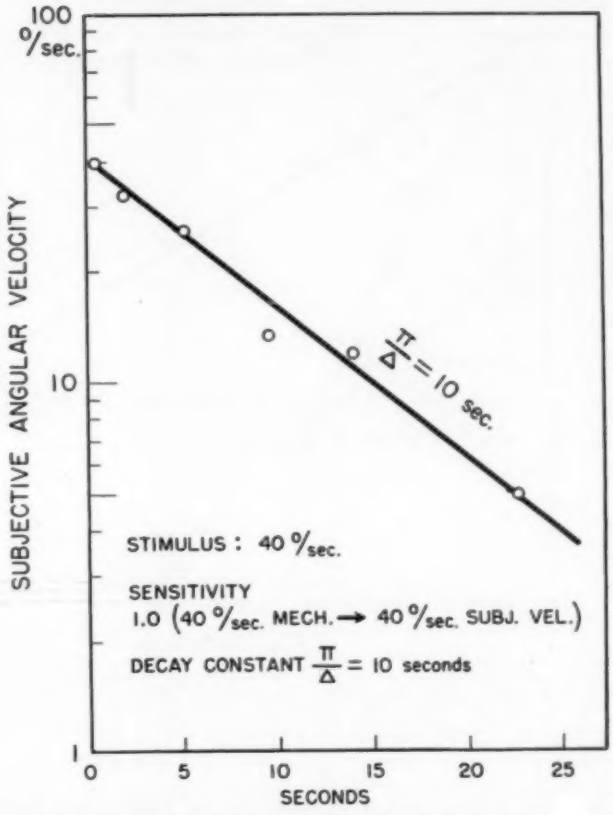

Fig. 5. Test subject is submitted to a clinical turning test: constant angular velocity of the chair before stopping is 40°/sec. Immediately after the stop the test subject feels himself rotating with such precision that he can tell whenever he has completed a full circle (360°). By checking these moments on a stop watch, started at the moment of the stopping of the chair, the average subjective velocity can be computed for each subjective full circle. These values have been plotted here. It appears that the subjective and the mechanical angular velocity are about equal. Not every test subject will show this equality. Some give higher, but usually the subjective value is lower than the mechanical one.

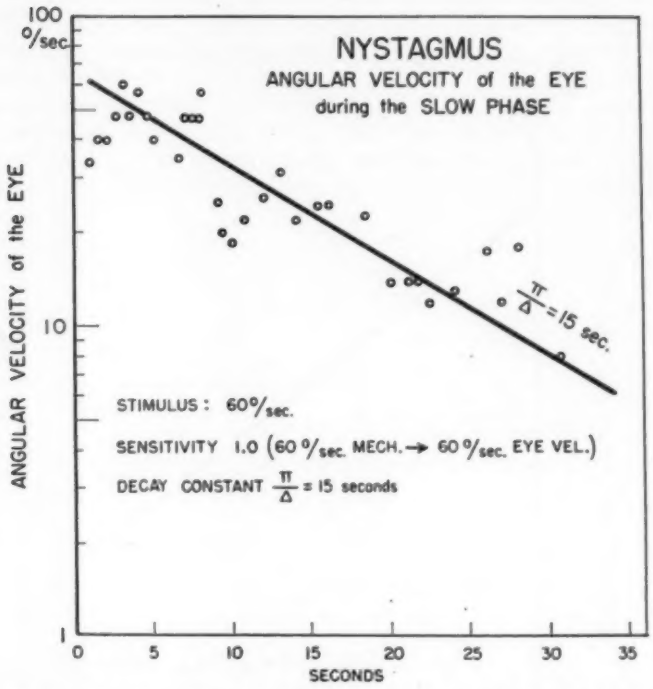

Fig. 6. The test subject is submitted to a clinical turning test with a constant angular velocity of 60°/sec. The nystagmus is recorded by the Schott procedure. The test subject is in total darkness. The angular velocity of the eye during the slow phase of the nystagmus is computed from the recording. It appears that the angular velocity of the eye just after the stop is about equal to the mechanical one. Usually an equality of only 80% is obtained.

of turning round and a nystagmus, of which the angular velocity of the eye during a slow stroke is related to the cupula deviation (van Egmond et al., 1951)[2]. It appears that an equality of 80 per cent between administered angular velocity, subjective (see Fig. 5) and eye velocity (see Fig. 6) is attained immediately after the stopping (Ruding, 1953)[10], but the sensation and the eye velocity have a different rate of decay. This is a conflict.

The return of the cupula and the associated phenomena,

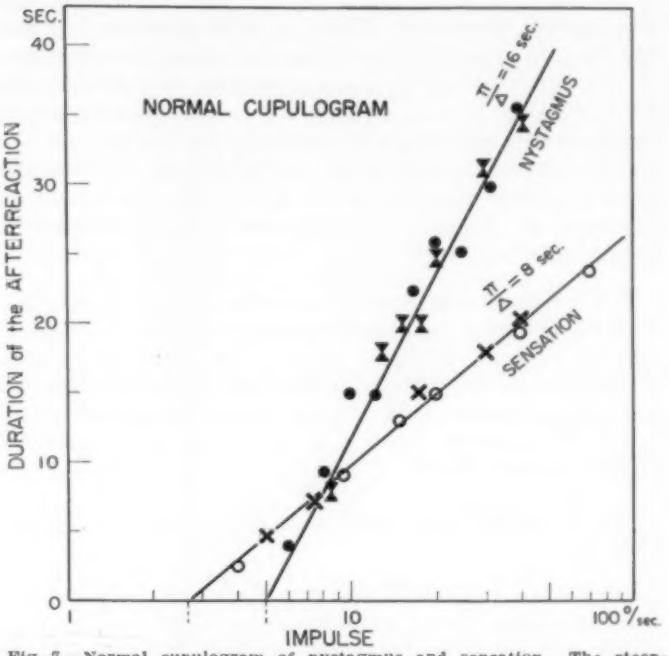

Fig. 7. Normal cupulogram of nystagmus and sensation. The steepness is different in the two graphs (i.e. 16 and 8 seconds). That the thresholds are different is of no importance. The nystagmuus threshold depends on the ability of the observer.

▼ ▲ Nystagmus	after turning clockwise	× sensation.
● Nystagmus	after turning counterclockwise	O sensation.

the sensation and the nystagmus, provide us with a means of determining the sensitivity, or better, the overall response of the vestibular system. The method of the clinical turning test applied as described is called "Cupulometry". Two curves relating the duration of the sensation and that of the nystagmus as a function of the impulse administered, contain the information required: the threshold and the rate of decay (see Fig. 7); but the conflict in the different rates of decay in one test subject remains to be explained. The discrepancy is too great to be ignored; $\frac{\pi}{\Delta} = 8$ seconds for the sensation and 16 seconds for the nystagmus.

We happened to try these experiments on a group of otherwise normal persons who had the same complaint; they all had "motion sickness" if submitted to a quasi-periodic change in vertical acceleration (deWit, 1953[12]; also van Egmond et al., 1954[3]). This is, of course, a reaction initiated by the otolith system and not by the semicircular canals; but their apparent lack of control of the reactions on linear movements extends as well to those on rotations. This group gave an identical rate of decay for sensation as for nystagmus, *i.e.* $\frac{\pi}{\Delta}$=20 seconds or more.

The low decay constants for sensation in the normal subject must be attributed to the inhibitional tendencies in the central nervous system (Krijger, 1954)[8]. As sensation is easier reached by the inhibiting action of the efferent fibers than the nystagmus, the latter having more the properties of a reflex, the curtailing action of the inhibition is stronger in the former (Groen, 1956)[6].

The anatomical data provide us with a calculated $\frac{\pi}{\Theta}$ value of 27 sec^{-1}.

The natural period of 6.3 seconds teaches us that $\frac{\Delta}{\Theta}$=1 sec^{-2}. so $\frac{\pi}{\Delta}$ calculated should be of the order of 25 seconds, which at last appears to be true; still we lacked the direct proof of the relation between phase difference and oscillation. It has been possible during the last months to establish this relation in our laboratory in some suitable test subjects, but not in all; Frederikse,[4] 1957. Again the difficulty of different behavior between test subjects mentioned before was encountered. Those results which could be considered as reliable and reproducible provide us with the data: $\frac{\Delta}{\Theta}$=1 sec^{-2}, $\frac{\pi}{\Theta}$=25 $seconds^{-1}$ and $\frac{\pi}{\Delta}$=25 seconds; so, in principle, the mechanical foundation of cupulometry is firmly established.

It has been said previously that the central nervous system tends to shorten the reactions to a non-physiological stimulus. This distortion is also present, but perhaps in a different way, when the test subject is submitted to an oscillation. During this test he is asked to observe on a screen the projection of a beam of light, which seems to move together with the

swing. The phase and the amplitude of the projector can be changed according to those of the swing. He is asked to manipulate two knobs (regulating phase difference and amplitude) until an exact coincidence between the image of the light and the feeling of the oscillating chair is attained. In this case there are a confusing number of interferences possible, so it is surprising that there are some persons who provide reliable data. The central nervous system does not like to solve riddles; as soon as there are some conflicting informations it tends to give preference to the most reliable one and this choice may be different in normal persons.

BIBLIOGRAPHY.

1. VAN EGMOND, A. A. J.; JONGKEES, L. B. W., and GROEN, J. J.: *Ned. Tydschr. Geneeskunde*, 87:1793, 1943. Also *J. Physiol.*, 110:1, 1949.

2. VAN EGMOND, A. A. J., and GROEN, J. J.: II *Congres O. R. L.*, Paris, 1951.

3. VAN EGMOND, A. A. J.; GROEN, J. J., and DE WIT, G.: *Int. Record of Med., G. P. Clinics*, 167:651, 1954.

4. FREDERIKSE, A. H.: Thesis, Utrecht, 1957 (to appear).

5. GROEN, J. J.; LOWENSTEIN, O., and VENDRIK, A. J. H.: *J. Physiol.*, 117:329, 1952.

6. GROEN, J. J.: Physics in Med. Biol., 1956.

7. HULK, J.: Thesis, Utrecht, 1949.

8. KRIJGER, M. W. W.: Thesis, Utrecht, 1954.

9. MACH, E.: "Grundlinien der Lehre von den Bewegungsempfindungen," Leipzig, 1875.

10. RUDING, J. H. H.: Int. Congres O. R. L., Amsterdam, 1953.

11. STEINHAUSEN, W.: *Pflueger Arch. d. ges. Physiol.*, 229:439, 1932.

12. DE WIT, G.: "Suppl. Acta Otol.," Stockholm, 108, 1953.

TRANS-SEPTAL-SPHENOID PITUITARY SURGERY.*

WALTER E. HECK, M.D.,
ROBERT C. MCNAUGHT, M.D.,
FRANCIS S. GREENSPAN, M.D.,
(By Invitation)
HARRY S. KAPLAN, M.D.,
(By Invitation).

San Francisco, Calif.

The trans-septal-sphenoid approach to the pituitary gland was developed by Oscar Hirsch in 1910. His work in this field will remain a monument in medicine.

INTRODUCTION.

This study on trans-septal-sphenoid pituitary surgery has several purposes. One is to review, consolidate, and clarify the many facets of the subject. Such facets as, for example, the anatomy involved and the surgical technique are of obvious interest to the otolaryngologist, and known to him in varying degrees; other facets, such as the histology of the pituitary and its tumors, the disturbed endocrinology resulting from pituitary tumors, and the various tests for these disturbances, are less well known, yet important, and especially deserve review and clarification.

A second purpose is to present a considerable experience with this type of surgery. Case reports include a series of 11 patients not previously reported in medical literature. In addition two techniques, which appear to be unique, are presented. One is the use of interstitial radon seeds, implanted by a trans-sphenoid approach. The other is the use of an implanted plastic tube for permanent drainage of a cystic pituitary tumor.

Third, the results of this series show that trans-septal-

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sphenoid pituitary surgery characteristically carries a very low mortality rate, and little morbidity; also it can accomplish some objectives unobtainable by the craniotomy approach. These facts deserve wider dissemination, so that all otolaryngologists, and more particularly specialists in other fields, *e.g.*, neurosurgeons, ophthalmologists, and endrocrinologists, will be more aware of what can be accomplished in the field of otolaryngology.

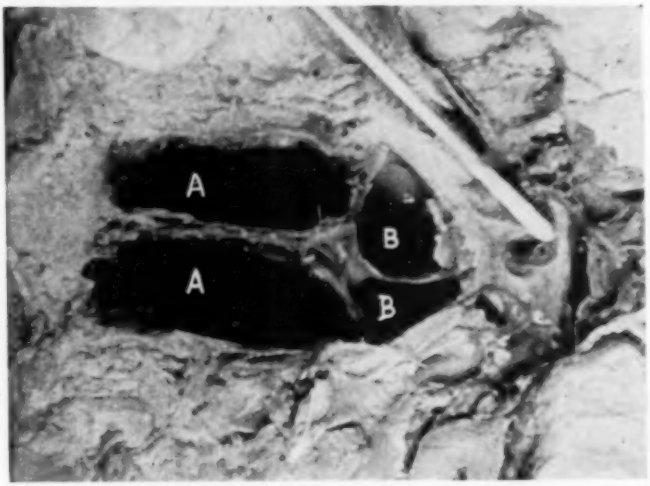


Fig. 1. Cadaver specimen, horizontal section through head at roof of nose, seen from above, showing: (A) nasal fossae; (B) sphenoid sinuses with inter-sinus partition between them; and pointer indicating infundibulum or stalk of the pituitary gland projecting through the opening in the dural diaphragm covering the sella turcica.

The historical aspects of this procedure have been described in great detail by Hirsch in his encyclopedic article in the *A.M.A. Archives of Otolaryngology* in 1952.[1] It includes 130 references. He cites how, at the turn of the century, intracranial approaches were first used, then various transsphenoidal approaches. These culminated in his trans-septal-sphenoidal approach.[2]

It is of interest that Cushing, combining a sublabial ap-

proach to the nasal septum devised by Halsted, used the trans-septal-sphenoid operation exclusively from 1910 to 1925, performing about 300 such pituitary operations.[3]

ANATOMY.

Since the upper posterior skeleton of the nasal septum is the vomer bone, submucous resection carried to this area is a direct approach to the rostrum (L. a beak) of the sphenoid

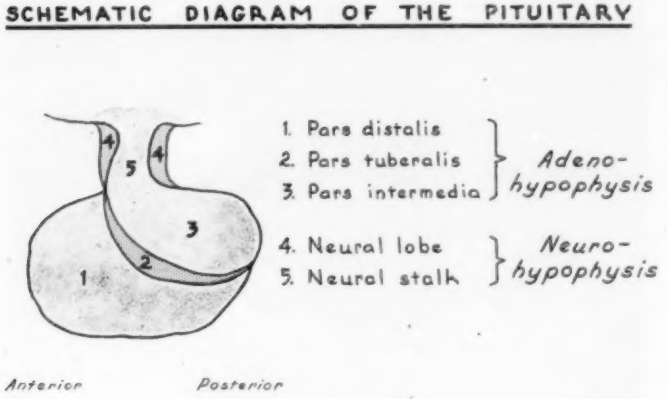


Fig. 2

bone. Removal of the rostrum provides an entrance into one or both sphenoid sinuses. Usually the intersinus partition is off the midline, to one side or the other. Removal of it creates a single sinus cavity, with the bulging postero-superior wall representing the antero-inferior floor of the sella turcica (Turkish saddle). Removal of this wall exposes the intrasellar periosteum, investing the pituitary gland.

Thus anatomically the trans-septal sphenoid approach to the pituitary is singularly direct.

The bean-shaped pituitary (L. glairy mucus) gland or

hypophysis (G. an undergrowth), lies in the osseus saddle, the sella turcica of the cranial base, and is connected to the floor of the third ventricle by a stalk, the infundibulum (L. a funnel), about one cm. in length. It is invested by a layer of periosteum lining, the sella turcica, which is continuous with dura mater. The dura forms a cover over the upper portion of the pituitary, closing the opening of the sella. This

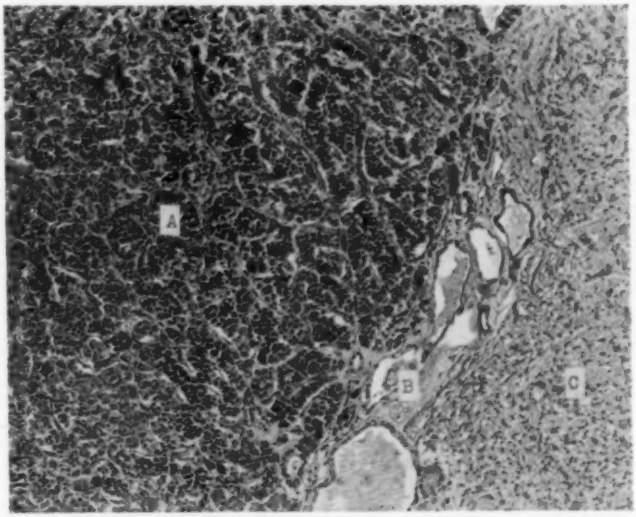


Fig. 3. Central section of normal pituitary showing: (A) pars distalis; (B) pars intermedia with its colloid-containing follicles; and (C) neural lobe. (X 112.)

cover or diaphragm of the sella has a central hole for the stalk (see Fig. 1).

On each side of the sella turcica and pituitary is the cavernous venous sinus, including the invested IIIrd, IVth, ophthalmic division of the Vth, and VIth cranial nerves. Above the pituitary, anterior to the stalk, is the optic chiasm. The optic tracts are to each side of the stalk.

The pituitary has two *primary* divisions. Older terminology, still used, refers to the anterior and posterior lobes.

More descriptive physiologically are the terms adenohypophysis, for the anterior lobe, and neurohypophysis, for the posterior lobe.[4]

The adenohypophysis is derived from the mucosa of the primitive buccal cavity, by the evagination of Rathke's pouch (craniopharyngeal duct). Thus it has the same anlage as have the teeth. The neurohypophysis is derived from the floor of the third ventricle.

The adenohypophysis is made up of three subdivisions, the pars distalis (pars anterior or pars glandularis), pars tuberalis, and pars intermedia. The neurohypophysis has two parts, the neural lobe (pars nervosa), and the neural

TABLE I (From Best and Taylor[4]).

Origin	Primary Division	Subdivisions
From primitive buccal cavity	Adenohypophysis (anterior lobe)	1. Pars distalis (pars anterior or pars glandularis.) 2. Pars tuberalis. 3. Pars intermedia.
From floor of third ventricle	Neurophypophysis (posterior lobe)	1. Neural lobe (pars nervosa). 2. Neural stalk.

stalk. This nomenclature is summarized in Table I, and illustrated in Fig. 2.

HISTOLOGY.

Taking these subdivisions in the order listed in Table I, the pars distalis is made up of three types of cells: *1.* Chromophobe (g. chroma, color, + phobos, fear) cells which are also known as chief cells, Gamma or reserve cells, small, with sparse, lightly staining, nongranular cytoplasm; and two types of chromophile (chroma, color, + philio, I love) cells. *2.* Eosinophiles (acidophil or Alpha cells); and *3.* basophiles (Beta cells). The eosinophiles are small, granular, and stain readily with red acid eosin dyes. The basophile is the largest of these three cells, with relatively coarse cytoplasmic granules, and greater affinity for the basic dyes such as the blue hematoxylin (see Figs. 3-4).

It is believed that the secreting chromophile cells are derived from the non-secreting chromophobe cells.

The pars distalis contains many vascular sinusoids.

The pars tuberalis may be considered an extension of the pars distalis upward around the hypophyseal stalk, and has a similar histology.

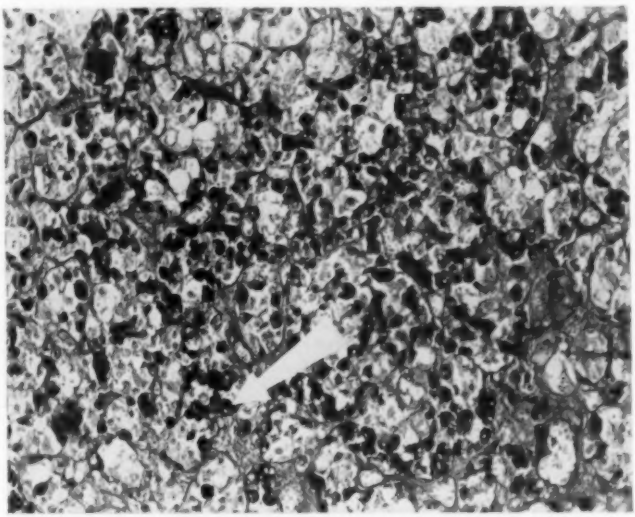


Fig. 4. Section of normal pars distalis (from Case No. 8) stained by the PAS technic (periodic acid Schiff) to distinguish the basophiles (arrow) by their granularity. (X 220.)

The pars intermedia, about 1 per cent of the anterior lobe, is characterized by colloid-containing follicles and cysts.

The neural lobe has five cellular components: *1.* Neuroglia (g. neuron, nerve, + glia, glue), the supporting protoplasmic network of this nervous tissue; *2.* Pituicytes, fusiform cells with several processes and brown pigment in their cytoplasm; *3.* Nerve fibers; *4.* Hyaline bodies (of Herring); and *5.* some basophile similar to those of the pars distalis,

some of which appear to degenerate and form large, round cells called oncocytes (g. onkos, tumor + kytos, cell).

The neural stalk again may be considered an extension upward, of the neural lobe, forming the greater part of the hypophyseal stalk, and has a similar histology.

PATHOLOGY: NEOPLASMS.

Although pathological conditions of the pituitary are

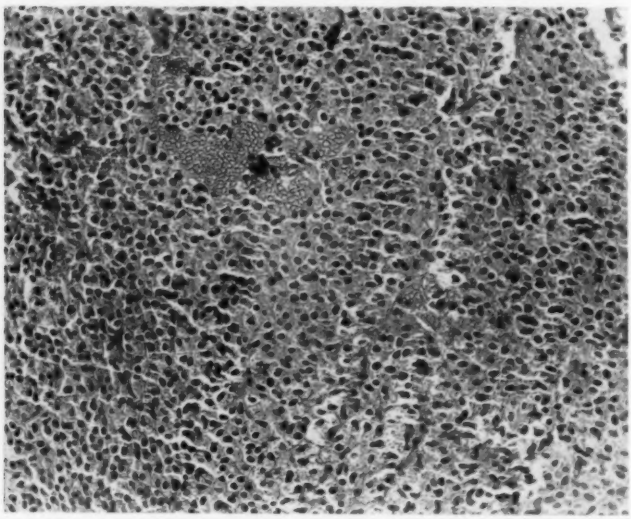


Fig. 5. Section of chromophobe adenoma from Case No. 5. (X 220.)

many,[5] those of particular interest to the surgeon are the neoplasms. Of these, adenomas are the most common. They usually arise from the pars distalis, and correspond to its three cell-types. The chromophobe adenomas (see Fig. 5) occur three times as frequently as the chromophile (eosinophilic and basophilic adenomas) types[6] (see Figs. 6-7). As noted above, the neurohypophysis has some basophile cells, and, therefore, basophilic adenomas may arise from this part of the pituitary.

The microscopic appearance of these tumors is, of course, characterized by the predominance of the particular cell type in the tumor mass. There may be several cell types—a mixed cell adenoma. The adenomas may be cystic.

The adenohypophysis derives from the stratified squamous epithelium of the primitive buccal cavity, just as do the teeth; therefore, a neoplasm histologically identical to the odonto-

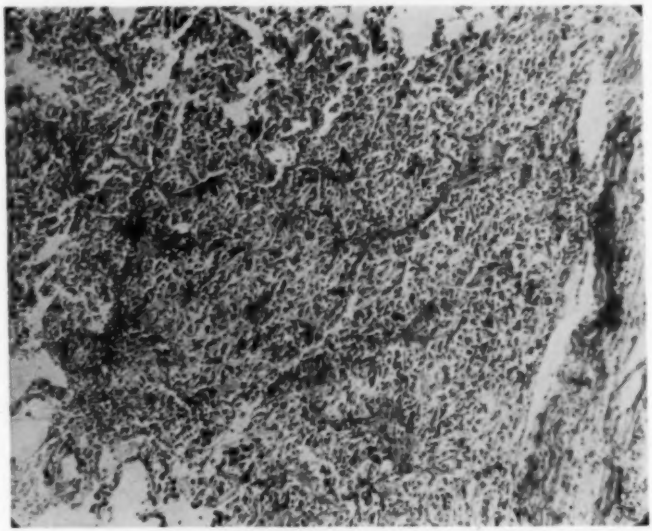


Fig. 6. Section of eosinophillic adenoma from Case No. 9. (X 112.)

genic adamantinoma[7] (ameloblastoma) may occur, called a craniopharyngioma, or Rathke's pouch tumor. It is second in frequency to the adenomas in neoplasms of the pituitary, and reportedly represents as much as 4.3 per cent of all intracranial neoplasms.

Microscopically the craniopharyngioma exhibits communicating strands of squamous epithelium, interspersed in a very loose stellate reticulum. That the craniopharyngioma and adamantinoma are identical is shown by the two micro-

photographs in Fig. 8. Craniopharyngiomas often show considerable calcification, which may be demonstrable on roentgenograms (see Fig. 19).

Although no reference to such a phenomenon has been found, it seems entirely plausible that actual tooth structures could form in a craniopharyngeal duct tumor; therefore,

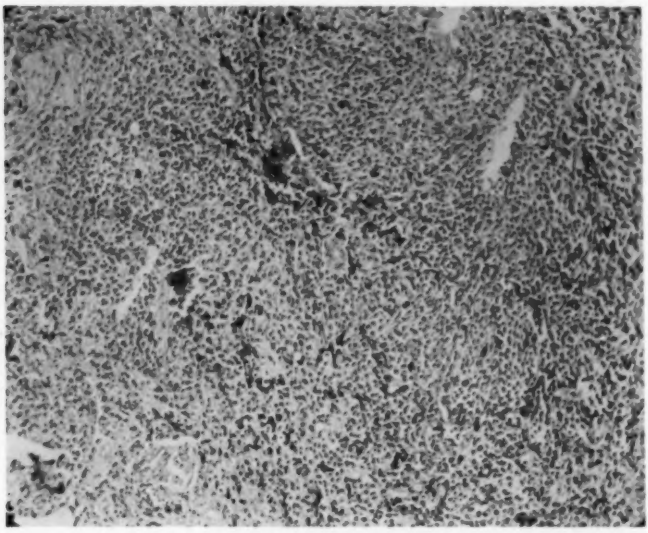
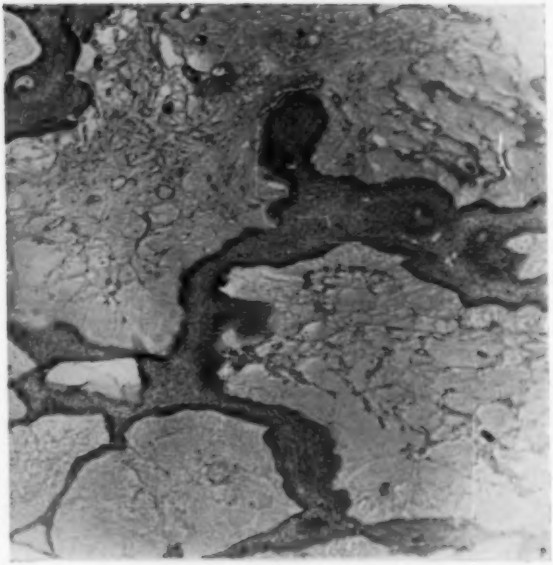


Fig. 7. Section of basophillic adenoma from Case No. 7. (X 112.)

prediction is ventured that a tooth-bearing tumor or cyst (odontoma or dentigerous cyst) of the pituitary area will some day be reported.

Many other neoplasms of the pituitary occur, including primary malignancies (see Fig. 9). Other tumors of this general area, *e.g.*, chordomas and aneurysms, must be kept in mind.

Tumors of the pituitary exert their effects on the patient in two ways: *1.* by endocrine dysfunctions; and *2.* by space-



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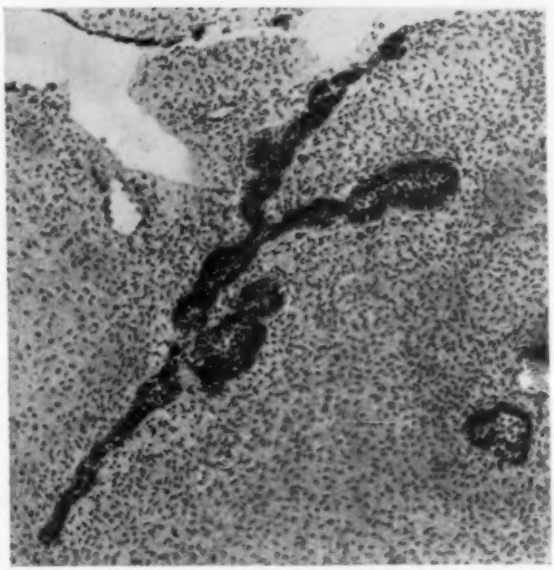

B

Fig. 8. (A) Section of craniopharyngioma (from Case No. 5) showing histologic similarity to (B) adamantinoma (from reference No. 7).

occupying enlargement and pressure upon adjacent important structures.

ENDOCRINOLOGY.

The adenohypophysis of the pituitary or "master" gland secretes a number of hormones, trophic (g. trophein, to nourish or nurture) in nature, effecting the development of

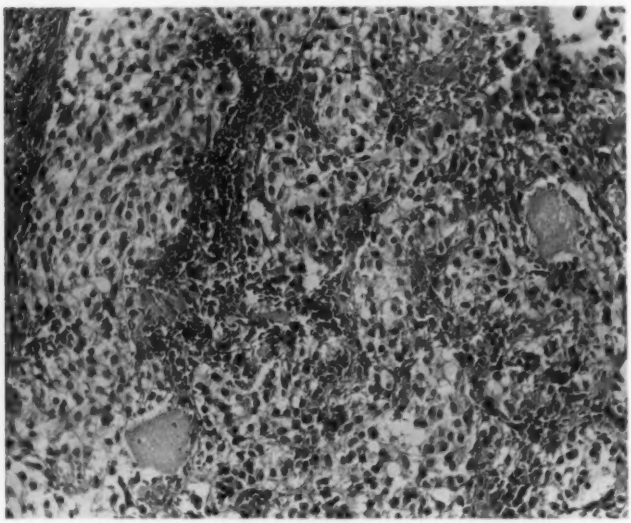

Fig. 9. Section of malignant tumor of pituitary, type undetermined, from Case No. 2. (X 220.)

other endocrines and thereby the individual. The following list includes those felt to be entities, and, in numbered parentheses, those not yet definite:

1. Somatotrophic (STH) (growth) hormone (secreted by the eosinophilic cells).

2. Thyrotrophic hormone (TSH).

3. Adrenocorticotrophic hormone (ACTH).

4. Luteinizing hormone
5. Follicle stimulating hormone } Gonadotrophic hormones.
6. Prolactin or lactogenic hormone.
(7) Ketogenic hormone.
(8) Insulin antagonizing or glycotrophic hormone.
(9) Diabetogenic hormone.

Fig. 10. Cadaver specimen, sagittal section through right nasal fossa, showing removal of rostrum of sphenoid by means of Sewall modification of Jansen-Middleton rongeur. Right nasal septal mucosa (A) has been folded down for purposes of demonstration; (B) pituitary; (C) sphenoid sinus; (D) nasal septum; and (E) external nose.

(10) Parathyrotrophic hormones.
(11) Pancreatrophic hormone.

Because of the diversity of these hormones, tumors and dysfunctions of the pituitary cause exceedingly varied patterns, difficult to classify. It would be nice to be able to place each case into a specific little niche, but this often cannot be done; however, certain fairly specific clinical types are recognized.

The chromophobe adenoma may present a clinical picture indicating a disorder of secretion of any of the above tabulated hormones; *e.g.*, there may be impairment of growth, loss of libido, diabetes, etc. With compression of the neurohypophysis, and the important neural hypothalamico-hypophyseal tract[8] (hypophyseo-diencephalic system), there may be diabetes insipidus or other manifestation of hypothalamic dysfunction; *e.g.*, Fröhlich's syndrome.

Fig. 11. Cadaver specimen, sagittal section through right nasal fossa, showing enlargement of opening in anterior face of sphenoid by means of Hajek rongeur.

The craniopharyngioma has similar endocrinological effects, the severity varying with the degree of impairment of the above tabulated hormones. The term "Simmonds' disease" has been associated with severe lack of function of the adenohypophysis, the so-called hypophyseal cachexia. The hypophyseal dwarf (Nanosomia = G. nanos, dwarf, + soma, body) pituitaria stops growing in childhood.

The eosinophilic adenomas are often associated with acromegaly (G. akron, extremity + megas, large) or Marie's

syndrome. If the dystrophy due to the tumor becomes manifest before the closure of epiphyseal lines, there may be simply giantism, rather than the grotesque appearance of the acromegalic.

Basophilic adenomas have long been associated with Cushing's disease (pituitary basophilism). Although they are

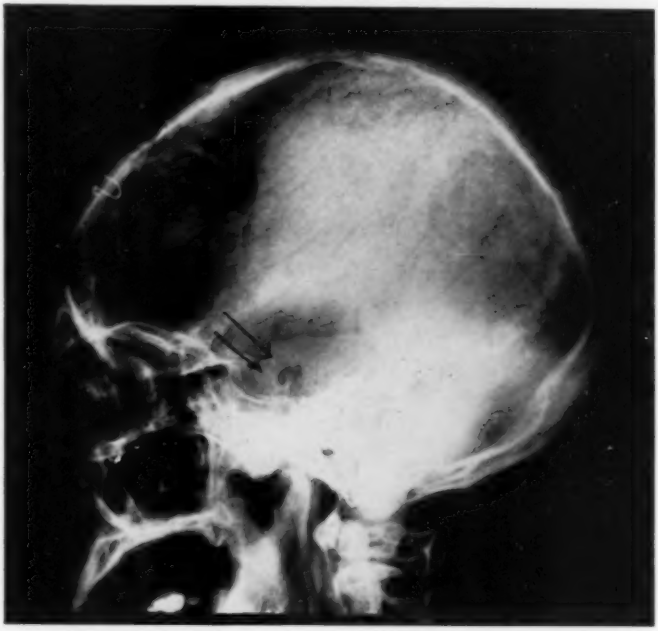

Fig. 12. Roentgenogram of Case No. 3 showing polyethylene plastic tube in place to drain cystic chromophobe adenoma. The outlines of the tube can be seen perpendicular to the arrows.

often concomitant, this syndrome appears to be primarily an adrenal cortex hyperplasia. A changed appearance of the basophiles often found in this condition has been termed Crooke's hyaline change. As mentioned above, basophiles are present in the neurohypophysis as well as the adenohypophysis, so that the adenoma may arise from either part.

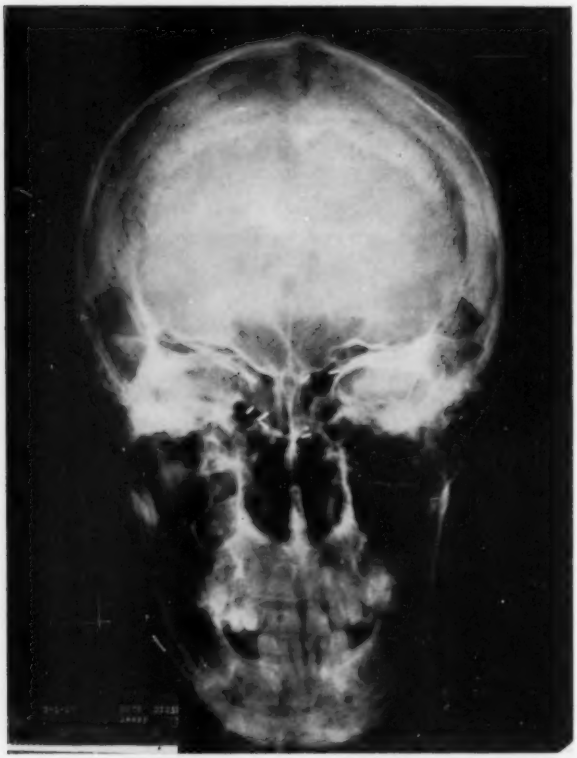

Fig. 13. Roentgenogram of Case No. 2 showing eight interstitial radon seeds in place in malignant tumor of pituitary. (For lateral view see Fig. 14.)

Again the resultant clinical picture depends upon the hormonal dystrophy.

Similarly, malignant tumors of the pituitary present a varied endocrinological picture, with the addition of manifestations of a malignant neoplasm as well as metastatic disease, if such is present.

In recent years there has been much study of the relationship of neoplastic growth to endocrine functions. Mammary

Fig. 14. Roentgenogram of Case No. 2 showing eight interstitial radon seeds in place in malignant tumor of pituitary. (For antero-posterior view see Fig. 13.)

carcinoma and prostatic carcinoma have been of particular interest since the demonstration that their growth can be altered by changes in hormonal environment.[9] Hypophysectomy has resulted in objective remissions in cases of breast carcinoma[10,11] and prostatic cancer, and the administration of beef pituitary growth hormone appeared to accelerate the growth of the tumor in one case of mammary carcinoma.[9] These facts suggest that a pituitary hormone or hormones may be important in the growth of some tumors.

EFFECTS OF EXPANDING TUMOR.

The second pathological manifestation of pituitary tumors is that of a locally expanding tumor in the sella turcica, tak-

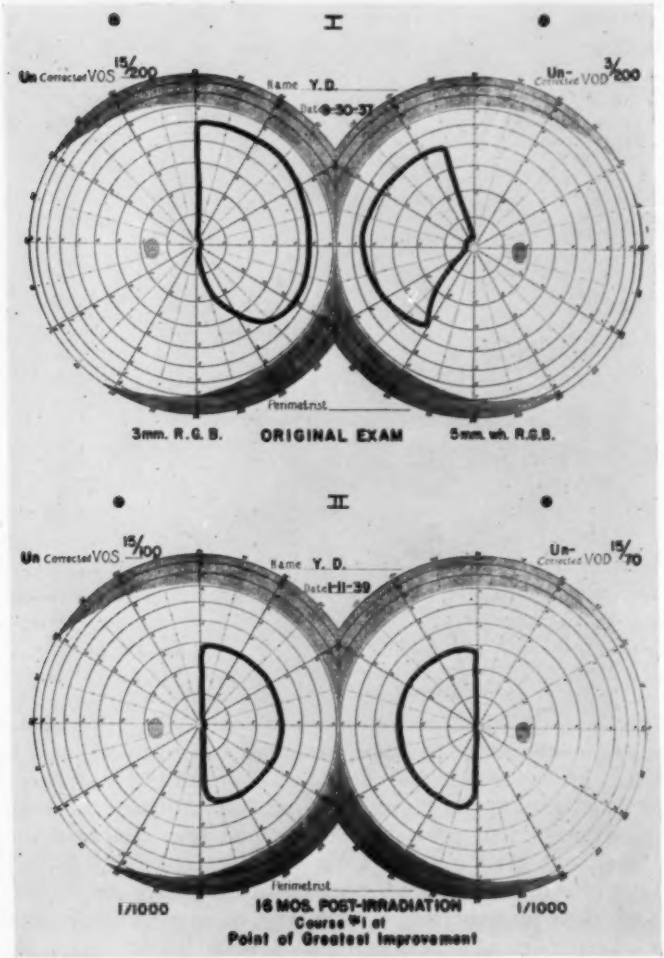

Fig. 15. Visual field charts of Case No. 3: I. Original examination in 1937; II. Examination in 1939 following first course of external irradiation, at time of maximum visual improvement. See Figs. 16, 17, and 18 for subsequent visual field examinations.

ing up intracranial space. The increased intracranial pressure may cause pressure headaches. Many important adjacent structures may be compressed, such as the optic tracts and optic chiasm, the brain stem with its vital centers, and the cerebrospinal fluid circulation system.

DIAGNOSIS.

The symptoms most commonly complained of are headache, a steady, pressure-type, and visual disturbances. The acromegalic may first note the skeletal changes, but often the lethargy, loss of libido and amnestic—confabulatory syndrome[12] are the disturbing factors to the patient and his family. Amenorrhea may be the chief complaint. Manifestations of diabetes mellitus, or diabetes insipidus, may be the presenting symptoms. One patient had convulsive seizures.

On examination of the eyes, the classical finding in pituitary tumor cases is a bitemporal hemianopsia (hemi, half + an-, priv., + opsis, vision) on visual field examination (see Fig. 15). The tumor as it grows, compresses the optic chiasm, containing the decussating fibers to the medial parts of the retinas; the resultant impairment of these fibers causes a loss of vision of the outer (temporal) visual fields. In recent study by Chamlin, et al., of 156 cases of pituitary adenomas and craniopharyngiomas, 86 per cent showed bitemporal hemianopsia.[13] Their series included 18 craniopharyngiomas, all of which showed bitemporal hemianopsia, as did 96 per cent of their 109 chromophobe adenomas. Some optic atrophy, as indicated by disc pallor, was present in 50 per cent of their cases, impaired visual acuity in 32 per cent, and extraocular muscle palsies in 5 per cent.

Neurological examination may reveal such extraocular muscle palsies, indicative of impairment of the IIIrd, IVth or VIth cranial nerves, and the optic nerve impairment (N. II). There may be increased intracranial pressure. An intracranial bruit may be heard on auscultation. Negative neurological findings may be very important in ruling out other intracranial tumors.

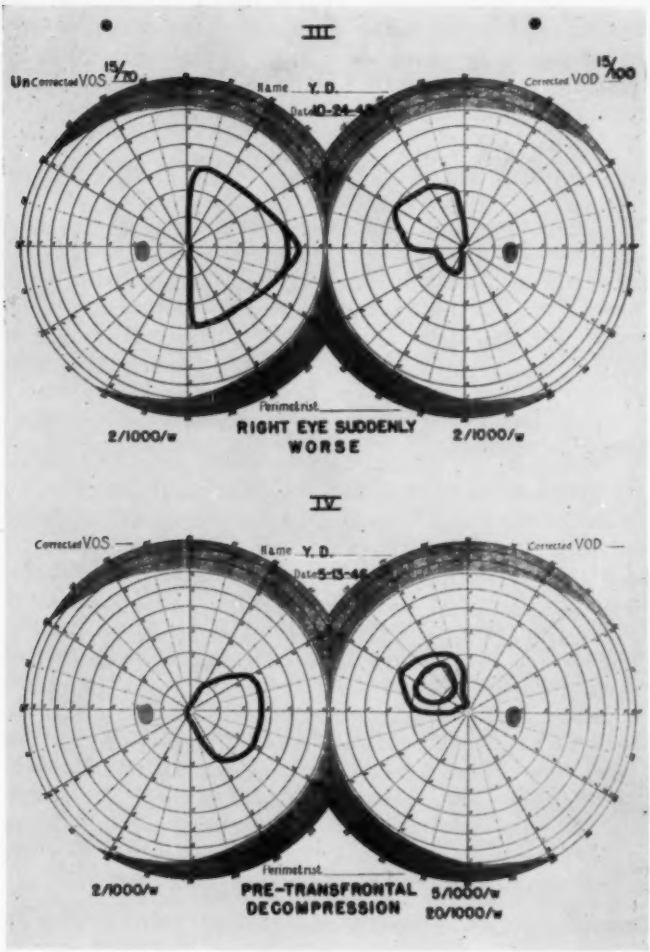

Fig. 16. Visual field charts of Case No. 3: III. Examination in 1945 when vision suddenly worse; IV. Examination in 1946 one month prior to trans-frontal operation. See Fig. 15 for preceding visual field examinations, and Figs. 17 and 18 for subsequent examinations.

Due to fixation of a crico-arytenoid joint, stretching of a vocal cord by the enlarged laryngeal box, or stretching of a recurrent laryngeal nerve, the acromegalic may have a fixed vocal cord(s).[14]

The endocrine status of the patient may be evaluated by comparison of his or her growth factors with standard growth-curve charts, and by laboratory tests which evaluate the function of the three major "target glands" of the adenohypophysis.[15] These "targets" include the gonads, the thyroid, and the adrenals. In addition, disease of the neurohypophysis may be present, indicated by symptoms of diabetes insipidus.

Gonadal function is clinically reflected by the menstrual history and libido. The role of the pituitary can be more directly evaluated by determining the pituitary gonadotrophin titer in the urine. To do this the urine is collected for 24 hours. The hormone-containing fraction is extracted and concentrated, and assayed in the immature mouse. One "mouse uterine unit" of pituitary gonadotrophin equals the amount of hormone required to stimulate sufficient estrogen to double the weight of the mouse uterus. Normal adult values are 5-50 m.u.u./24 hr. Unfortunately, there are no simple, practical assay methods for estrogens and androgens.

As for the thyroid, there is no direct biological test for the thyroid stimulating hormone (TSH) as yet available. There are, however, several tests of spontaneous functions of the thyroid. The basal metabolic rate (BMR), of course, falls as thyroid function decreases; conversely the blood cholesterol rises as thyroid function decreases (normal 113-230 mgm./ml. plasma cholesterol). An index of the circulating thyroxin is the amount of serum protein bound iodine (PBI). Normal is 5-8 micrograms/100 ml. This will decrease in quantity with decreased TSH. Care must be taken to be sure the patient is not on iodine therapy, or has taken other extraneous iodine (*e.g.*, in the course of some roentgenographic study). In addition, the uptake of radioactive iodine, I^{131}, will be impaired if advanced pituitary disease has reduced the thyroid function. The normal uptake is

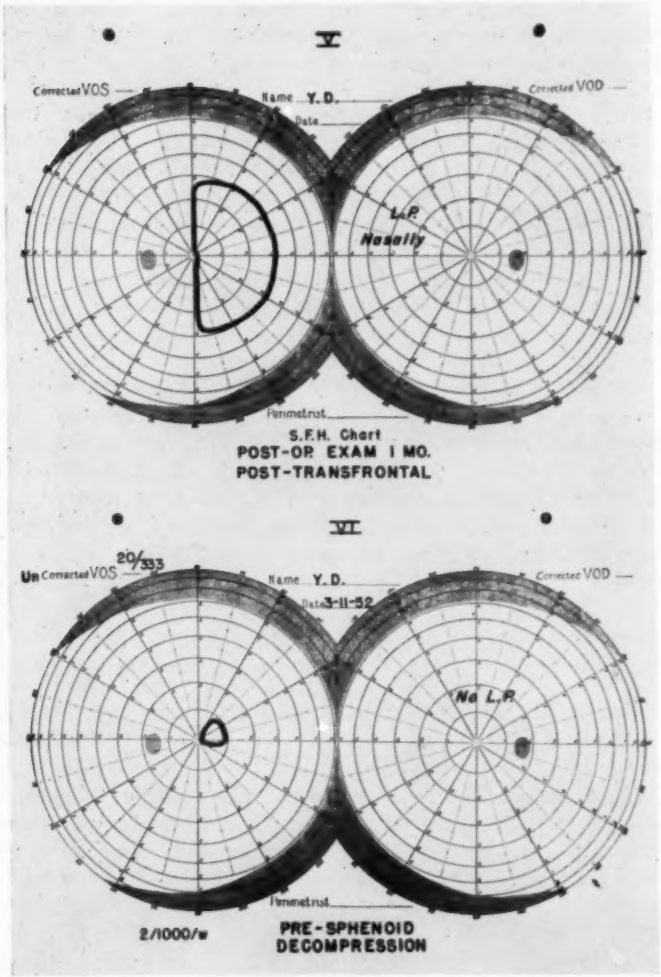

Fig. 17. Visual field charts of Case No. 3: V. Examination in 1946 one month after trans-frontal operation; VI. Examination in 1952 one month before trans-septal-sphenoid surgery. See Figs. 15 and 16 for preceding visual field examinations, and Fig. 18 for subsequent examinations.

15-40 per cent in 24 hours. To determine whether an impaired uptake is actually due to a pituitary deficiency, and not to a primary thyroid disorder, thyroid stimulating hormone (TSH) may be injected as a test. If 10 units is given daily for two days the I^{131} uptake will usually be doubled, given a thyroid gland capable of responding.

Adrenal function may be checked by a number of tests. One such is the water test (Robinson-Power-Kepler). In the absence of compounds E (Cortisone) and F (hydrocortisone) the kidneys are unable to excrete a water load; therefore, if a patient is given one-and-a-half quarts of water, an output of 1200 ml. in the next five hours will indicate adequate corticoids; under 800 ml. output indicates a deficiency. The blood electrolytes such as sodium (normal serum Na 135-142 mEq./L.) and potassium (normal 4-5 mEq./L.) may be present in abnormal concentration with adrenal disease; they are usually normal in patients with pituitary disease.

The *sine que non* of adrenal studies is the determination of the adrenal steroid metabolites in the urine. These include the 17 ketosteroids (normal for women 5-18 mgm./24 hrs.; for men 10-23 mgm./24 hrs., 30 per cent of which is from the male gonads), 17 hydroxycorticoids (11 oxysteroids) (normal for women 4-9 mgm./24 hrs.; for men 4-11 mgm./24 hrs.), and recently aldosterone. The latter appears to be independent of control by the adenohypophysis; therefore it is normal in patients with pituitary disease.[16] The former two, however, precursors of compounds E and F, fall to low levels with decreased adrenocorticotrophic hormone (ACTH).

The response to ACTH may be used to distinguish between pituitary lack or adrenal insufficiency. One test is to give an infusion of 25 units of ACTH over a course of 6 to 8 hours; if the adrenals are functioning normally there will be a marked rise in 17 ketosteroids (usually doubles) and 17 hydroxycorticoids (usually triples), while the eosinophiles disappear from the circulating blood. Or ACTH gel may be given, 40 units twice daily for two days, as the stimulus. If there is long-standing pituitary disease present, these responses may be delayed in appearance.[17]

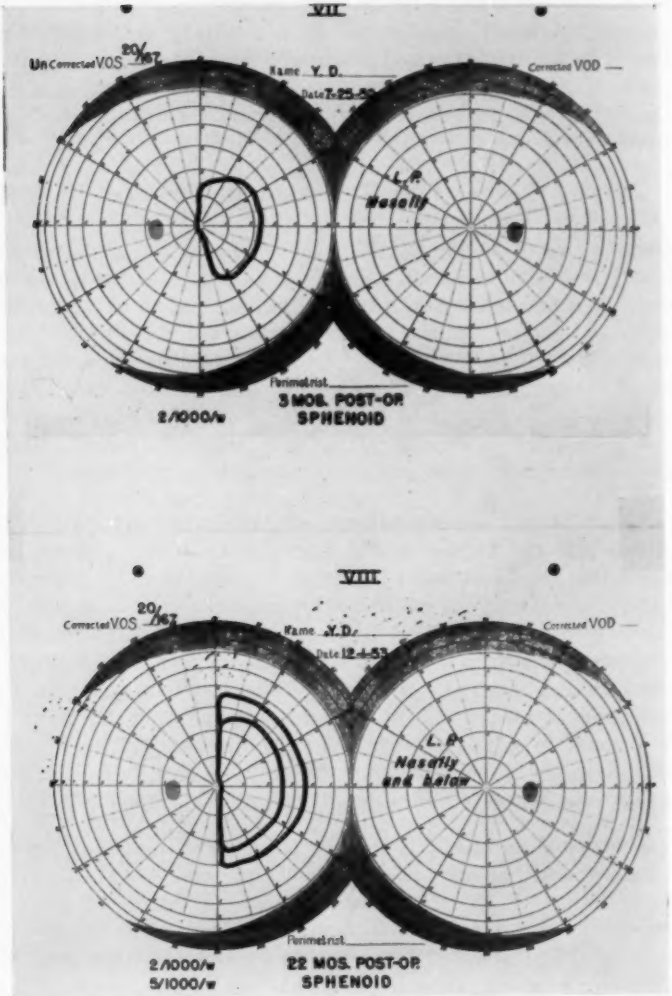

Fig. 18. Visual field charts of Case No. 3: VII. Examination in 1952 three months after trans-septal-sphenoid surgery; VIII. Examination in 1953 twenty-two months after trans-septal-sphenoid surgery. See Figs. 15, 16, and 17 for preceding visual field examinations.

Diabetes insipidus, indicative of dysfunction of the neurohypophysis, is manifested by a fluid intake of over 5-6 L./24 hours, and an output of an equal volume of urine of fixed, low specific gravity.

On roentgenological examination the ballooned sella turcica is typical of the pituitary tumor. The antero-inferior wall,

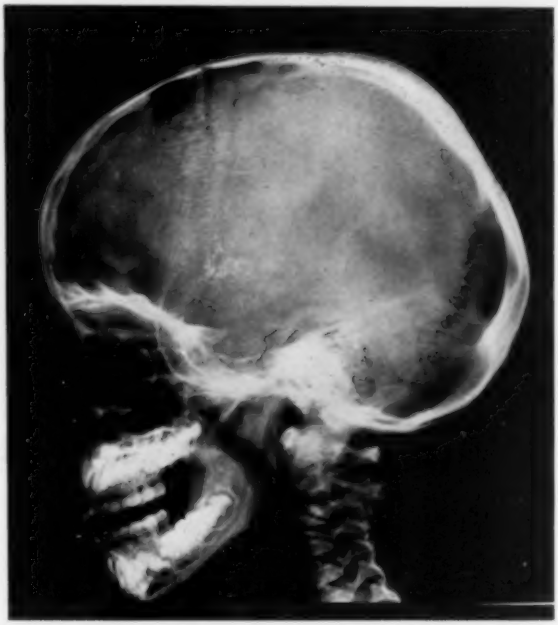

Fig. 19. Preoperative roentgenogram of Case No. 5 showing calcification in craniopharyngioma to be high above sella turcica. (See Fig. 20 for postoperative roentgenogram.)

the postero-superior wall of the sphenoid sinus, may actually be destroyed. Calcium deposits, intra- or suprasellar, may be demonstrated, characteristic of craniopharyngioma. Although the anterior and posterior clinoid processes may be eroded by an adenoma, with carcinoma there may be widespread bone destruction, with obliteration of the foramina ovale and spinosum on the basal skull views.

Fig. 20. Postoperative roentgenogram of Case No. 5 showing calcified area in craniopharyngioma has dropped markedly and is now in sella turcica area, indicating that extensive subtotal removal of tumor has been accomplished. (See Fig. 19 for preoperative roentgenogram.)

Special studies may be required, such as ventriculograms and arteriograms. The latter particularly may demonstrate the presence of an aneurysm or arteriovenous fistula, and should be done before any surgery is attempted if there is any question of the diagnosis.

HORMONE MANAGEMENT.

As experience with pituitary surgery and hypophysectomy increases, it is increasingly apparent that subtotal and total removal or destruction of the "master gland" is not as disturbing to the patient as once supposed. Required steroid therapy and other replacement therapy is surprisingly little.

Generally Cortisone (compound E, or one of the newer equivalents, hydroxycortisone (compound F), prednisone (Delta Cortisone) or prednisolone (Delta hydrocortisone) is given pre-operatively, *e.g.*, Cortisone 50 mgm. I.M. every four hours for 2 to 6 doses, and continued postoperatively until oral therapy can be given. This dosage is gradually tapered to a maintenance dose of 25 mgm. once or twice daily.

Thyroid deficiency, with symptoms of myxedema, usually does not appear, if at all, until four to eight weeks post-operatively. Replacement is readily attained by 60-120 mgm. dessicated thyroid daily.

The polyuria of diabetes insipidus may be controlled by nasal insufflations of posterior pituitary powder two or three times daily, or by Pitressin[R], I.M.

Appropriate sex hormones may also be indicated, *e.g.*, estrogens or androgens.

ANESTHESIA.

For obvious reasons a general anesthesia is required in children in order to accomplish a trans-septal-sphenoid pituitary operation. Care should be taken to have an endotracheal tube in place, preferably with an inflatable cuff, and in addition, a hypopharyngeal wet gauze pack should be placed, to prevent blood going down the patulous esophagus into the stomach, causing postoperative nausea and vomiting.

In adults topical and local anesthesia is much more satisfactory. A sedative is given the night before surgery. Moderately heavy premedication of a barbiturate, morphine sulphate and scopolamine hydrobromide is given pre-operatively, the dosages varying with the weight and apprehension of the patient. The patient's face is prepared and draped aseptically. After spraying the nasal fossae with a vasoconstrictor, *e.g.*, 1 per cent ephedrine, cotton pledgets moistened, but not dripping, with a solution of equal parts of 10 per cent cocaine and 1:1000 epinephrine HC1 are placed in each nasal fossae. Usually three pledgets are used in each side. After

placing these, six more pledgets are similarly prepared. Then the first set of pledgets are removed. Usually now the space within the nose is seen to be markedly increased. The second set of pledgets can then be much more strategically placed, especially back against the face of the sphenoid.

After removal of the second set of cotton pledgets, a standard local anesthetic such as 1 per cent procaine or lidocaine HCl (Xylocaine^{R}) is injected, starting at the face of the sphenoid subperiosteally, using a long needle, then forward on the septum subperiosteally and subperichondrally, not only for greater anesthesia but also to elevate the mucosa hydraulically from the sphenoid and septum, to aid the mechanical elevation of the operation.

TECHNIQUE.

When using the customary topical and local anesthesia, it is usually found easiest to operate with the patient in a semi-sitting position. The head mirror provides the best illumination, permitting co-axial vision within the reflected beam, through the keyhole-like nostril into the nasal fossae and far back into the depth of the operative field. Use of a high-watt bulb (*e.g.* 100 watts) as the source of the light gives brilliant illumination. Headlights of various kinds can and have been used, but the factor of a parallax always must be overcome.

Having completed the anesthesia, a submucous resection of the nasal septum (SMR) is performed. If the septum has a deviation, a routine anterior incision may be used, thus incidentally straightening the patient's septum in the course of the operation. If the septum is straight, the incision can be conveniently made about half way back in the nose, preferably on the left side of the septum for the right-handed operator. The septal mucosa posterior to the incision is then elevated from the septal skeleton, back to the rostum of the sphenoid. A mistake the novice makes is to elevate the mucosa only enough to make a small tunnel. Instead, it is most important to elevate the *entire* septal mucosa posterior to the incision, and its reflections onto the roof

and floor of the nasal fossa. Thus the entire flap can be pushed laterally, against the lateral wall of the nose. After breaking through the septal skeleton the opposite septal mucosa is similarly elevated and the exposed septal skeleton removed, as in the routine SMR. Complete elevation of the mucosal flaps posteriorly, from the face of the sphenoid, cannot be stressed too much. Thus they are both pushed to the respective lateral walls, and, since the bony septal skeleton has been removed, an "envelope," or tunnel, as wide as the *combined* nasal fossae gives excellent exposure of the face of the sphenoid.

This elevated mucosa contains the many nasal mucosal blood vessels; therefore if it is not torn, no bleeding occurs, and the operation becomes practically bloodless. Care not to tear the mucosa is especially important on the face of the sphenoid, where the large septal branch from the sphenopalatine artery crosses to reach the septum.

These steps in the back of the nose are facilitated by use of a long bladed nasal speculum.

Removal of the bony rostrum is accomplished by using a Sewall modification of the Jansen-Middleton rongeur (see Fig. 10). This opens one or both of the sphenoid sinuses, depending upon the position of the intersinus partition of the sphenoid. Usually the partition is off to one side of the midline, and at first only one sphenoid sinus is entered. As the opening is enlarged, by means of Hajek and Kerrison rongeurs, both cavities are entered (see Fig. 11). This opening in the face of the sphenoid can usually be made to the size of a nickel (21 mm.). The intersinus partition and its mucosa is then excised, using ring punches and such instruments as the Wilde cup nasal forceps. The two sphenoid sinuses are thus made into one large cavity.

The mucosa of the sloping, rounded posterior wall of this cavity is then incised in an upside-down U pattern, and this flap of mucosa folded downward by its inferior pedicle-hinge on the floor of the cavity for later use. This flap may have two parts, if the intersinus partition happened to be in the midline before it was removed.

Initial opening through the bony posterior wall, into the sella turcica, is made by rotating a long narrow gouge manually, like a dental drill. Other instruments have also been used. This exposes the sellar periosteum (dura). The opening is then enlarged by means of the Hajek and Kerrison rongeurs. Usually this opening can be made to the size of a dime (18 mm.).

The preceding step may be entirely unnecessary if a pituitary tumor is present which has broken through the posterior wall, from the sella into the sphenoid. If the bony wall was intact, its removal has accomplished decompression of the tumor.

The distance from the nostril to the sella, usually 9.5 cm., is then measured and recorded for later use.

A long aspiration needle can then be inserted into the tumor, to check whether it is cystic.

Biopsy can be taken by means of a cup forceps, biting directly into the encapsulating periosteum-dura. Subtotal removal of the tumor may be carried out. If the operation is not for pituitary tumor, but rather for hypophysectomy, it can be accomplished.

If a cystic tumor is encountered, it can be opened and drained. Then the previously described U-shaped mucosal flap can be reflected into the cyst cavity, in an attempt to create permanent drainage. In addition, a piece of plastic tubing may be placed with one end in the cyst, the other end supported by the floor of the sphenoid sinus (see Fig. 12). (A search of the medical literature did not reveal any previous record of this latter step).

The nasal mucosa is then replaced, and held in place by packing, such as vaseline gauze. This is removed about 48 hours postoperatively.

Usually adult patients, done under topical and local anesthesia, are up and about postoperatively the day of surgery. Prophylactic antibiotic therapy is given for five days.

On examination of the healed interior of the nose no ab-

normality can be detected unless pressure on the septum or face of the sphenoid reveals the flexibility of the mucosa.

Hirsch has stressed the use of postoperative irradiation by means of locally applied radium to prevent regrowth of remaining tumor and possibly achieve its shrinkage.[18] Actually interstitial radiation may be achieved by the insertion of radon seeds, by means of long needle carriers, into the remaining tumor (see Figs. 13-14). (Again, a search of the medical literature did not reveal any previous record of this technique.) This may be done anytime after the mucosa has healed (*e.g.* 10 days postoperatively). Topical anesthesia is again used to anesthetize the nasal fossae (after preparation and draping of the patient's face), not only for the anesthesia but also to reduce the nasal congestion to permit good exposure of the interior of the nose. The radon carrier needles are inserted directly through the intact mucosa of the face of the sphenoid, into the tumor. This is possible because the bony skeleton is now absent from the nickel-size area in the face of the sphenoid, and the dime-size opening in the backwall of the sphenoid. The previously noted distance from nostril to tumor is used to determine how far into the tumor a seed is being placed. By directing the needle carriers—sometimes even from one nasal fossa, through the sphenoid cavity, to the opposite side of the tumor—the seeds are distributed in the tumor. It is helpful to have an observer behind the patient, and one beside the patient, to help site the direction of the needle carriers. This technique is illustrated in a motion picture describing the trans-septal-sphenoid operation, by Heck and McNaught.[19] Radioactive cobalt has also been used interstitially.[20]

INDICATIONS.

The principle indications for the trans-septal-sphenoid operation are: *1.* Rapidly-progressing loss of vision; *2.* incapacitating head pressure symptoms; *3.* hypophysectomy for palliation of hormone-labile carcinoma, *e.g.*, mammary or prostatic carcinoma; *4.* convulsive seizures due to focal irritation; *5.* amennorhea; *6.* need for local application or interstitial radiation (*e.g.* maximal external irradiation al-

ready given); *7.* for diagnostic biopsy; and *8.* inability to tolerate craniotomy, or refusal to permit it.

OBJECTIVES.

Many objectives may be attained by this operation. They include:

1. Decompression (permanent).

2. Cyst drainage (permanent).

3. Diagnostic biopsy.

4. Subtotal removal of tumor.

5. Local irradiation, applied or interstitial.

6. Hypophysectomy.

It should be emphasized that, unlike the decompression obtained by the craniotomy approach, the decompression accomplished by the trans-septal-sphenoid approach can be a permanent one. Closure of the craniotomy restores the solid bony box containing the tumor; upon its regrowth and expansion the same situation exists as before the operation. With the trans-septal-sphenoid approach, however, the tumor, as it regrows and expands, can bulge downward into the sphenoid cavity, and even push the flexible mucosa (across the face of the sphenoid) ahead of it to expand into the nasopharynx.

Similarly, the cyst drainage may be permanent, the cyst fluid spilling into the sphenoid cavity, then through the tiny sphenoid ostiae in the nasal mucosa into the nasopharynx, to be swallowed. A plastic tube drain may be implanted to keep the cyst open.

CASE REPORTS.

The present Stanford Otorhinolaryngology Division series of trans-septal-sphenoid pituitary operations consists of eleven cases. It is of interest, however, that as long ago as 1915 Sewall, then head of this Division, reported on a case done by this method.[21] Two of our eleven patients were reoperated,

one of whom had implantation of interstitial irradiation, and three others had interstitial irradiation through the intact mucosa, making a total of 16 operative procedures.

Case 1. D.B., male, age 22, was operated on Sept. 22, 1950, by the trans-septal-sphenoid approach, indicated as a diagnostic procedure. He had a left temporal visual field defect, and diabetes insipidus. Roentgenograms showed an enlarged sella. Upon exposure of the sellar periosteum, the sellar contents was needled in three places to determine whether a cystic pituitary lesion was present. No cyst was encountered by the aspirating needle. Neurosurgical consultation in the operating room advised against biopsy. Postoperative course was uneventful; he was dismissed from the hospital, ambulatory, on the second postoperative day. Eight months later the patient died, and autopsy revealed an ectopic pinealoma of the floor of the third ventricle.

Case 2. O.C., male, age 37 at the time of surgery in 1952, had a history of meningovascular syphilis treated with Neoarsphenamine*, and of a cerebro-vascular accident in 1941, which left him with some residual right hemiparesis. In January, 1952, his chief complaint was head pain described as a sensation of pressure. Skull roentgenograms revealed marked destruction of the body and sella of the sphenoid. Eye examination revealed a left central scotoma (G. skotoma, darkness) and vision in the right eye of 20/67, left 20/167. Right carotid arteriograms on Jan. 16, 1952, were normal. Blood and spinal fluid Wasserman were negative; B.M.R. was minus 25.2 per cent; 17 ketosteroids excretion was 7.6 mgm./24 hrs. (normal 10-23 mgm./24 hrs.).

A trans-septal-sphenoid operation was performed on Feb. 2, 1952, and biopsy revealed a malignant tumor of the anterior pituitary (see Fig. 9). Except for a right sixth cranial nerve palsy postoperatively, the postoperative course was uneventful. The patient was up and about on Feb. 5, 1952, and left the hospital on the seventh postoperative day. On March 7, 1952, he returned, and eight 1 millicurie radon seeds were placed into the malignant tumor in the pattern of the corners of a cube (see Figs. 13-14). This interstitial radiation gave a calculated dose of 3,000*r*. (Welsh,[22] using radium, advised a maximum of 1000 mgm. hours.) He then received, in addition, a course of external irradiation, in his home community, sufficient to cause epilation. Later there was regrowth of his hair. Thereafter he was remarkably well until January, 1955, when he developed a transient cerebrospinal fluid rhinorrhea. This fluid contained "sugar" reducing substance of 77 mgm. per cent. The rhinorrhea stopped spontaneously three months later. At that time also, surprisingly enough, his visual fields were found to be intact, without scotomata, and vision in the right eye 20/30, left 20/30. Laboratory tests in July, 1955, included 17 ketosteroid excretion of 6 mgm./24 hrs., 17 hydroxycorticoids of 1 mgm./24 hrs., (normal 4-11 mgm./24 hrs.), sodium 144 mgm./100 ml. (normal 330 mgm./100 ml.) and potassium 43 mgm./100 ml. (normal 16-22 mgm./100 ml.). He also complained of impotence. Because this suggested panhypopituitarism he was started on a regimen of methyl testosterone 10 mgm., bid.

He is alive and active, with this substitution therapy, four and one-half years postoperatively.

Case 3. Y.D., male, age 46, when operated upon in 1952, first complained of impaired vision in 1937. At that time he was found to have a bitemporal hemianopsia, (Figs. 15, 16, 17, and 18 show this and subsequent visual field charts of this patient); vision 5/200 right, 15/200 left, with a marked-

*Registered trademark.

ly enlarged sella on roentgenogram; therefore, he received a course of external X-ray to the pituitary tumor of 2800*r*. In August, 1937, his right visual field was larger, but the left more constricted. He did not return for follow-up as often as was desired. In 1945 he received an additional 1560*r*. of external X-ray, and neurosurgery was advised. On June 20, 1946, a right frontal craniotomy was performed with subtotal removal of a bluish, walnut-sized cystic chromophobe adenoma. He was heard from occasionally during the next few years, complaining only of absence of libido and impaired vision. When seen in the spring of 1952, however, the patient was almost blind. He had only a tiny visual field in the left eye, and not even light preception in the right. He carried a white cane for guidance.

A thorough preoperative endocrine work-up was done, including the demonstration of subnormal urinary gonadotropins. The Stanford Tumor Board advised trans-septal-sphenoid pituitary surgery to decompress the cystic tumor and, if possible, establish permanent drainage of the cyst. He was operated on April 15, 1952, and a polyethylene plastic tube placed to permanently drain the cystic tumor (see Fig. 12). The patient was discharged from the hospital two days later, after removal of the nasal packing. One week postoperatively he already commented on improved vision. Three months postoperatively he was found to have an improved visual field in the left eye and light perception in the right. Now, four years postoperatively, he returns for checkups, having discarded the white cane and reads the newspaper while in the waiting room.

Case 4. B.E., male, age 46, at the time of surgery, had been having seizures since 1948, only partially controlled by Dilantin*. Although there was no visual field loss demonstrable in 1948, the sella turcica was enlarged and eroded on roentgenological examination. It was felt at that time, however, as noted in his chart, that whatever was causing the marked erosion could not be helped surgically. Ventriculogram studies were done July 30, 1951. A year later, on June 17, 1952, after a thorough endocrinological study had shown a status of endocrine balance, a trans-septal-sphenoid decompression was performed. A polyethylene tube drain was placed into the cystic chromophobe adenoma (see Fig. 5). Preoperatively he had been somewhat lethargic, and this persisted postoperatively; his seizures were markedly less frequent, requiring much less drug therapy. Otherwise he was generally well when last seen on Jan. 9, 1954.

Case 5. R.S., a little boy age 7, when first operated upon by us in 1953, had begun to complain of headaches when four years of age. With this he developed a weakness of the left side of his face, became too weak to walk, and his left eye turned outward and became fixed (exotropia -G. exo, outward, + trope, turn), with a dilated pupil. Roentgenograms at another hospital, including ventriculograms, revealed a large calcium containing tumor of the pituitary area, a craniopharyngioma. External irradiation, 4000*r*. was given to the area. Thereafter he became stronger for a while, but then the headaches returned, and the left eye became blind.

In March of 1953 ventriculograms were repeated, and on March 19, 1953, his first trans-septal-sphenoid pituitary operation was performed, under general anesthesia, using an endotracheal tube and a hypopharyngeal pack. A large quantity of the cystic tumor was removed, a quantity of tissue about equivalent to the size of a hen's egg. Microscopically it was a typical craniopharyngioma (see Fig. 8). A plastic tube drain was left in place. He was ambulatory on the day after surgery. Postoperative roentgenograms showed a marked drop of the calcified areas,

*Registered trademark.

previously above the sella (see Fig. 19), into the sella (see Fig. 20), serving as an index of how much tumor had been removed. Thereafter he was well enough to attend school regularly.

Seven months later he again began having severe headaches, and roentgenograms showed the tumor had regrown to its original size, the calcium deposits being again raised out of the sella. On Oct. 28, 1953, a second subtotal removal was performed, by the trans-septal-sphenoid approach. The septal mucosal flaps were surprisingly easy to separate, and the operation was easier than the primary procedure. The plastic tube drain was removed, and a metal clip placed as a marker for roentgenograms. One week later 10 one millicurie radon seeds were inserted into the tumor as interstitial irradiation. Even though craniopharyngiomas do not respond to irradiation, the fact that he had improved years before after external irradiation prompted the use of these seeds. He again returned to school. About ten months later he began having nosebleeds. The tumor was growing down into and filling the nasopharynx. He received an additional 1100*r*. of external irradiation, and this apparently caused enough fibrosis to stop the bleeding. In the hope that a more curative result might be obtained, some experimental tumor chemotherapy was tried, but the drugs merely created a severe anemia problem.

In May, 1955, the tumor became so large as to fill the nasopharynx and right nasal fossa, pushing the flexible nasal septum to the left. Subtotal removal was again accomplished, this time by a right Caldwell-Luc approach, removing the tumor and right lateral wall of the nose in a fashion sometimes used for juvenile fibromas.[23] A Fox post-nasal balloon was used as packing in the nasopharynx.

It was felt that the trans-septal-sphenoid operations had provided a means of permanent decompression of the tumor. It could expand into the nasopharynx, where relatively little harm is done, rather than intracranially, where great damage and death would ensue. One year later, in May, 1956, the tumor had again reexpanded, even presenting in the right nasal vestibule; therefore, the fourth subtotal removal was performed, the operation this time by a trans-palatal approach.

Although theoretically such subtotal removal can be continued indefinitely, yet it is hoped that this child's tumor may spontaneously "burn itself out". Meanwhile his parents are grateful for the excellent palliation he gets from surgery. He is again in school. He happens to have a twin brother, who is much larger than the patient, demonstrating the lack of growth hormone in the patient.

Case 6. M.C., female, age 53, had acromegaly with diabetes and marked retinitis. Because of severe pressure headaches a trans-septal-sphenoid decompression of her chromophobe adenoma was performed on March 26, 1953. She left the hospital one week later, ambulatory. A month later she felt much improved, and was able to do her own urinalyses, something she could not do preoperatively.

Seven months later pressure symptoms recurred. It was planned to use interstitial irradiation. At surgery, however, it was found that the bone had completely grown across the face of the sphenoid under the mucosa, closing the operative dehiscence; therefore, it was necessary to repeat the operation, and then insert the six radon seeds. This regrowth of bone was unique, perhaps associated with her acromegaly.

Now three years postoperatively she is alive, but remains in a hospital, on a regimen of multiple replacement drugs.

Case 7. G.F., female, age 31, complained of amenorrhea and headache, and was referred from her home in Alaska when roentgenograms

revealed a greatly enlarged sella. No abnormalities were found on examination of the eyes. After endocrinological studies were made, a trans-septal-sphenoid pituitary operation was performed on July 17, 1953, the biopsy showing a basophillic adenoma (see Fig. 7). Two weeks later six radon seeds were placed into the tumor at a second operation. Three years postoperatively she still has amenorrhea, but feels fine, and has lost 30 pounds of excess weight. She now has a right altitudinal hemianopia, and may require further treatment in the future.

Case 8. C.M., female, age 55, had had a hysterectomy and oophorectomy in 1945 at 47 years of age. She developed a carcinoma of the right breast two years later (1947), for which she received X-ray therapy. The following year, 1948, she had a right radical mastectomy, in which 17 of 17 regional nodes were found to contain metastases. In 1952 her vocal cords became fixed in the adducted position, requiring tracheotomy. Additional X-ray therapy was given in 1953, and she also received testosterone. By September, 1953, she was very ill, with advanced mammary carcinoma uncontrolled locally and with disseminate metastases. Roentgenograms showed a normal sella turcica. The Stanford Tumor Board felt that hypophysectomy might give some palliation. Although the patient was told she might not survive the operation, she agreed to it. On September 14, 1953, she was taken to the operating room, unconscious and obviously moribund. Although death was imminent, the hypophysectomy was performed, using the trans-septal-sphenoid approach (see Fig. 4). A rather vigorous attempt was made to insure completely removing the pituitary. The patient lived for 36 hours postoperatively, never regaining consciousness. Autopsy revealed the widespread metastatic adenocarcinoma, and that the pituitary grossly had been completely removed. The right posterior clinical process had been fractured and there was subarachnoid hemorrhage about the brain stem. Uterus and adnexae were absent. The other many findings are not pertinent.

Case 9. M.Y., female, age 36, an acromegalic with enlarged sella on roentgenogram, had biopsy and decompression of her pituitary eosinophilic adenoma (see Fig. 6) by the trans-septal-sphenoid approach on March 9, 1954. Surgery had been suggested by the Endocrinology Division because of her marked acromegalic syndrome of asthenia, headaches, vomiting, decreased libido, amenorrhea, and blurred vision. A temporal defect and central scotoma was present in the visual field of the right eye. She left the hospital on the eighth postoperative day. Two months postoperatively all her symptoms were greatly improved.

A program of hormone substitution was started. Five months later she again began to have menstrual periods. Two years postoperatively she is in relatively good health, taking 5 mgm. hydrocortisone bid and thyroid 120 mgm. daily, and caring for her home and three children.

Case 10. O.R., female, age 34, had an expanding sellar tumor on roentgenograms, and was developing visual impairment. She refused any type of diagnostic or therapeutic craniotomy procedure. On Feb. 21, 1956, biopsy was obtained by a trans-septal-sphenoid approach, to which she was amenable. The postoperative course was typically uneventful. Microscopic examination of the tissue by frozen section technique suggested chromophobe adenoma, but fixed sections were interpreted as normal pituitary tissue. She received a course of external irradiation to the pituitary area, and her vision is now improved; however she is still being carefully observed as to the possibility of some other neurological tumor or disease unrelated to the pituitary (see Case 1).

Case 11. D.H., female, age 56, complained of impaired vision for three months. She was found to have diabetes insipidus, destruction of the

posterior clinoid processes and dorsum sella on roentgenograms, and a bitemporal hemianopia. Her neurosurgeon felt the diagnosis could be best established by biopsy by the trans-septal-sphenoid approach. This was done on Aug. 13, 1956. The tumor was found within the sphenoid sinus, having already broken through the posterior sinus wall. Microscopic examination of the fixed tissue revealed the tumor was a squamous cell carcinoma. X-ray therapy by means of a 6,000,000 volt linear electron accelerator was inaugurated.

MORTALITY AND MORBIDITY.

These case reports emphasize the low mortality and low postoperative morbidity associated with trans-septal-sphenoid pituitary surgery. The only death in our series was the patient moribund at the time of operation, who had widespread mammary carcinoma, on whom palliative hypophysectomy was performed; she should be excluded from the series of local pituitary tumor cases.

Hirsch reports an over-all mortality rate of only 6 per cent for his impressive series of 413 cases, including many from the pre-antibiotic era and his early pioneer cases.[17] A representative mortality figure for the craniotomy approach, in chromophobe adenomas alone is 11.2 per cent, by Grant[24]; in craniopharyngiomas it is even higher.

Postoperative morbidity is usually minimal. As reported above, several cases were well enough to leave the hospital two days after surgery. This is in sharp contrast to the usual craniotomy case.

RESULTS.

Thus this series consists of eleven patients, five male including one child, and six females. In the ten patients with primary pituitary tumors the results of trans-septal-sphenoid surgery have all been good, in some cases excitingly so. The one patient who died was moribund with widespread metastatic carcinoma at the time of operation; complete hypophysectomy *was* accomplished in this case.

CONCLUSIONS.

The trans-septal-sphenoid pituitary operation is an excellent procedure for several reasons. Characteristically

there is practically no postoperative morbidity, and it carries a very low mortality rate.

It can accomplish permanent decompression of an expanding pituitary tumor, by permitting the tumor to expand postoperatively into the nasopharynx, where little harm is done, rather than upward against the vital cranial contents. The craniotomy approach cannot accomplish this.

Similarly, permanent drainage of a cystic tumor can be accomplished, by permitting the interior of the cyst to drain into the sphenoid cavity, with or without a plastic tube drain, and then into the nasopharynx. Again this is unique.

This surgical approach creates a means of convenient implantation of radon seeds for interstitial irradiation. Hypophysectomy can also be accomplished.

Because the craniopharyngioma has the same anlage as the adamantinoma, it is predicted that some day a Rathke's pouch tumor will be found containing tooth structures, a dentigerous cyst or odontoma.

BIBLIOGRAPHY.

1. Hirsch, Oscar: Symptoms and Treatment of Pituitary Tumors. *A.M.A. Arch. Otolaryngol.*, 55:268-306, March, 1952.

2. Furstenberg, A. C., and Blatt, I. M.: Trans-Sphenoid Approach to the Pituitary Gland. The Laryngoscope, 65:420-430, June, 1955.

3. Scarff, J. E.: Fifty Years of Neurosurgery, 1905-1955. *Surg. Gynec., and Obst.*, 100:417-513, Nov., 1955.

4. Best, C. H., and Taylor, N. B.: "The Physiological Basis of Medical Practice," 1330 pp., 5th Ed. Williams & Wilkins Co., Baltimore, 1950.

5. Anderson, W. A. D.: "Pathology,"2nd Ed., 1393 pp. C. V. Mosby Co., St. Louis, 1953.

6. Nurnberger, J. I., and Korey, S. R.: "Pituitary Chromophobe Adenomas," 282 pp. Springer Pub. Co., Inc., New York, 1953.

7. Heck, W. E.: Odontogenic Tumors and Cysts. The Laryngoscope, 62:1097-1111, Oct., 1952.

8. "The Pituitary Gland," 764 pp. Association for Research in Nervous and Mental Disease, XVII. Williams & Wilkins Co., Baltimore, 1938.

9. Pearson, O. H., Ray, B. S., Harrold, C. C., West, C. D., Li, M. C., MacLean, J .P., and Lippsett, M. B.: Hypophysectomy in the Treatment of Advanced Cancer. *Trans. A. A. Physicians*, 68:101-111, 1955.

10. Luft, Rolf, and Olivecrona, Herbert: Hypophysectomy in Man. *Cancer*, 8:261-270, March-April, 1955.

11. Pearson, O. H., Li, M. C., MacLean, J. P., Lipsett, M. B., and West,

C. D.: Management of Metastatic Mammary Carcinoma. *J.A.M.A.*, 159:1701-1704, Dec. 31, 1955.

12. White, J. C., and Cobb, Stanley: Psychological Changes Associated with Giant Pituitary Neoplasms. *Arch. Neurol. and Psych.*, 74:383-396, Oct., 1955.

13. Chamlin, Max, Davidoff, L. M., and Feiring, E. H.: Ophthalmic Changes Produced by Pituitary Tumors. *Am. Jour. Ophthal.*, 40:353-368, 1955.

14. Grotting, J. J., and Pemberton, J. deJ.: Fixation of Vocal Cords in Acromegaly. *A.M.A. Arch. Otolaryngol.*, 53:608-617, 1950.

15. Greenspan, F. S.: Personal communication.

16. Leutscher, J. A., and Axelrad, B. J.: Sodium-Retaining Corticoid in the Urine of Normal Children and Adults and of Patients with Hipoadrenalism or Hypopituitarism. *Jour. Clin. Endocrin. and Metab.*, 14:1086-1089, Sept., 1954.

17. Escamilla, R. F.: "Laboratory Aids in Endocrine Diagnosis," 131 pp. Charles C. Thomas, Springfield, Ill., 1954.

18. Hirsch, Oscar: Pituitary Tumors, A Borderland Between Cranial and Trans-Sphenoidal Surgery. *New Eng. Jour. Med.*, 254:937-939, 1956.

19. Heck, W. E., and McNaught, R. C.: Motion picture, "The Trans-Septal-Sphenoid Operation," 16-mm. Color, Silent, 1000 ft., obtainable on loan from the Division of Otorhinolaryngology, Stanford University School of Medicine.

20. Baron, S. H.: Personal communication.

21. Sewall, E. C.: Operation on the Hypophysis according to Hirsch's Method. *J.A.M.A.*, 65:681, 1915.

22. Walsh, T. F.: Trans-Septal-Sphenoid Approach with Implantation of Radium to Pituitary Fossa as Treatment for Adenomatous Tumors of the Pituitary Gland. The Laryngoscope, 53:724-735, 1943.

23. Heck, W. E., and McNaught, R. C.: The Transpalatal Approach for Juvenile Fibroma. *Calif. Med.*, 75:430-431.

24. Grant, F. C.: Surgical Experience with Tumors of the Pituitary Gland. *J.A.M.A.*, 136:668-672, 1948.

CHROMOPHOBE ADENOMA OF THE NASOPHARYNX.

JOSE AQ. AQUINO, M.D.,*
FRANCIS CHRISTOPH, M.D.,†
and
EDWARD MURRAY, M.D.,‡
Newark, N. J.

Chromophobe adenoma of the nasopharynx is still a rarity. A survey of the literature reveals that the first report was that of Harvey Cushing, in his book entitled "The Pituitary Body and Its Diseases," published in 1912, and in which he mentioned three cases. Henderson, in studying 338 of Cushing's pituitary tumor series, notes that only eight had nasopharyngeal extension, and none entered the nasal cavity. Shea, however, reported a case of bilateral tumor of the nasal septum. Bailey and Cutler also noted that one case of their three had invaded the nasal cavity.

The presence of a pituitary adenoma in the nasopharynx is explained in two ways, namely:

1. It may reach this cavity by direct extension from the sella turcica. This is the most common pathway.

2. It possibly could arise from congenitally displaced cells along the course of Rathke's pouch in its development as an outpocketing of the buccal mucosa just anterior to the oral plate. This extends upward and backward to make contact with the diencephalic portion, partially wrapping itself around the latter as it extends up into the base of the brain.

CLINICAL FEATURES.

Aside from the general symptoms of headache, bitemporal

*Chief Resident, Head and Neck Department, Newark Eye and Ear Infirmary, and Martland Medical Center.
†Associate Surgeon, Otolaryngology Dept., Newark Eye and Ear Infirmary, Newark, N. J.
‡Attending Pathologist, Newark Eye and Ear Infirmary, Newark, N. J.
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hemianopsia and others due to excess of glandular secretions, the patient may complain of the following:

1. Nasal obstruction.

2. Blood-tinged sputum.

3. Rhinorrhea (especially those with previous "polypectomy" and this may be due to cerebrospinal leakage).

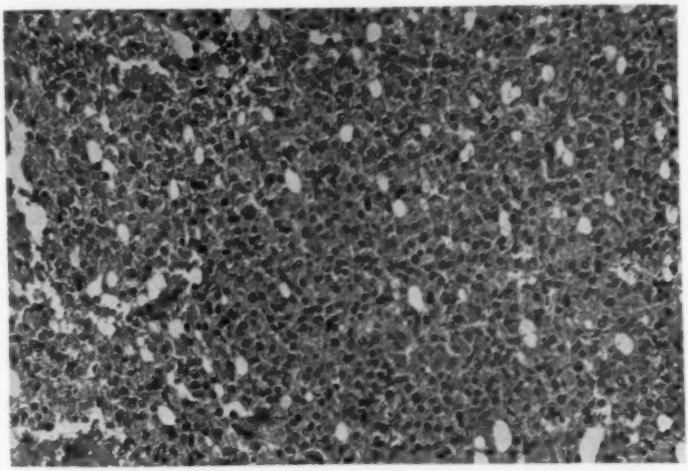

Fig. 1. Tumor cells are arranged roughly in radial manner around the fine vascular sinuses.

4. Troublesome epistaxis with excessive bleeding.

5. Intermittent unexpected discharge of mucus into the pharynx.

CASE REPORT.

A 44-year-old Caucasian male consulted a physician, with the chief complaint of "stuffiness of the nose."

The present illness began with a severe frontal headache, slight epistaxis and associated nausea and vomiting. Examination at this time revealed the presence of slight hemorrhage in the posterior pharynx and right anterior nares. There was also a slight injection of the conjunctiva of the right eye, proptosis of the right eye and poor pupillary response to light in the right eye. There was no ciliary tenderness. Vision at that time was 20/200 O.D. and 20/20 O.S.

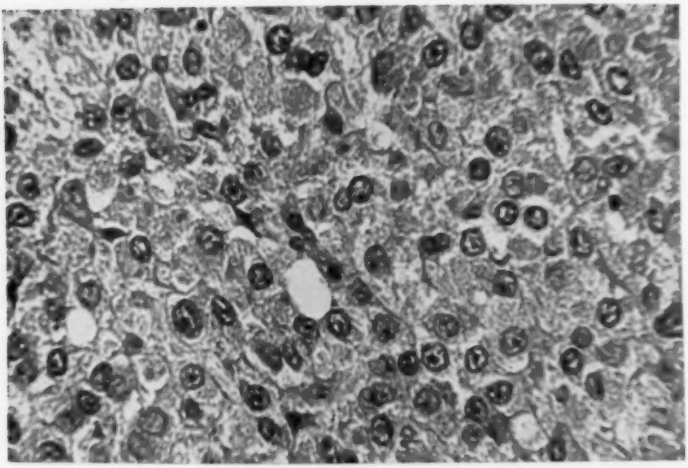

Fig. 2. High power magnification showing the cells which are polygonal or slightly elongated with delicate cytoplasm which is free of granules and the nuclei which are oval and darkly stained.

Fig. 3. Lateral view of the skull showing the classical ballooning of the sella turcica.

A diagnosis of acute sinusitis with complicating cavernous sinus thrombosis was made, and the patient was sent to the hospital for treatment. While in the hospital, there was tenderness over the right ethmoid area, proptosis of the right eye and oozing of fluid through the right nares. He developed a fever (101.4°F.) and nuchal rigidity. He was then treated as a case of meningitis with chemotherapy and antibiotics. He recovered from the "meningitis," and examination of the nasopharynx revealed a granular mass in the right side measuring about 1 cm. in diameter. X-rays of the sinuses showed "no bony changes."

Polypectomy was done. Histological examination of the specimen showed "polypoid rhinitis with exuberance of plasma cells". Patient was discharged after an uneventful recovery.

Ten months later, he returned to the office complaining of fronto-temporal headache of several days' duration. Transillumination showed cloudiness of the right frontal sinus. There was also slight tenderness. He was then treated with antibiotics and analgesics. Subsequent visits to the office for "stuffy nose" revealed no nasal tumor. Following skin tests for allergy, he was treated with antihistamines, anti-allergy injections, and advised to stop smoking. Nineteen months later, a large

Fig. 4. Basal view of the skull showing the difference in densities in the regions of the sphenoid sinuses.

mass was found in the middle meatal region. A polypectomy was done, followed by cauterization of the turbinates. Two years later, a mass was again found in the nasopharynx. Polypectomy was done and the specimen was sent to the pathologist, who made a diagnosis of "chromophobe adenoma of the pituitary."

X-rays of the skull then showed the classical ballooning of the sella turcica. A final diagnosis of chromophobe adenoma of the pituitary with extension into the nasopharynx was made. Patient referred for X-ray treatment.

COMMENTS.

In retrospect, the diagnosis should have been made in the very beginning, as the presenting symptoms of severe frontal headache with epistaxis, conjunctival injection and proptosis, associated with the poorly reacting pupil and a vision of 20/200 would indicate an intracerebral lesion. The proptosis and the conjunctival injection may be explained by a local venous stasis phenomenon.

When the patient developed "meningitis," it must have coincided with the rupture of the diaphragma sellae or floor of the sella turcica. The polyps previously removed were probably not part of the pathology, as they may develop independently of the adenoma "polyp" as was seen in one of the cases of Kay, et al.

SUMMARY.

A case of chromophobe adenoma of the nasopharynx is reported in addition to those in the literature. When removing polyps from the nasopharynx, it is worthwhile considering the presence of chromophobe adenoma, especially in those cases where symptoms persist.

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BIBLIOGRAPHY.

1. Bailey, Percival: "Intracranial Tumors"—2nd Ed. Charles C. Thomas, Springfield, Ill., 1948.

2. Cushing, Harvey: "The Pituitary Body and Its Diseases." Lippincott, Philadelphia, Pa., 1912.

3. Henderson, W. R.: The Pituitary Adenomata; a Follow-Up Study of the Surgical Results in 338 Cases, by Dr. Harvey Cushing. *Brit. Jour. Surg.*, 26:811-921, 1938-1939.

4. Kay, S.; Lees, J. K., and Stout, A. P.: Pituitary Chromophobe Tumors of the Nasal Cavity. *Cancer*, 3:695-704, July, 1950.

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President: Dr. Jose Gros, Havana, Cuba.
Executive Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa., U. S. A.
Meeting: Sixth Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
Time and Place: Brazil, 1958.

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Secretary-Treasurer: Lt. Col. Sanley H. Bear, M.C., 3810th USAF Hospital, Maxwell AFB, Alabama.
Time and place of meeting: October 15, 1957, Palmer House, Chicago, Ill.

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Meeting: Roanoke, Virginia, December 6 and 7, 1957.

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